DUPLICATE

TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

TWENTY-SIXTH SESSION

HELD AT THE RED LION INN, STOCKBRIDGE, MASS.
MAY 26, 27 AND 28, 1914

EDITED BY

LINNAEUS EDFORD LA FÉTRA, M.D.

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PRESIDENTS

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M.D.
1906. A. Jacobi, M.D.
1907. B. K. Rachford, M.D.
1908. C. G. Kerley, M.D.
1909. Charles P. Putnam, M.D.
1910. David L. Edsall, M.D.
1911. Henry Dwight Chapin, M.D.
1912. Walter Lester Carr, M.D.
1913. John Lovett Morse, M.D.
1914. Samuel McC. Hamill, M.D.
1915. George N. Acker, M.D.

OFFICERS, 1914

President ..... Samuel McC. Hamill, M.D.
Vice-President ..... Matthias Nicoll, Jr., M.D.
Secretary ..... Samuel S. Adams, M.D.
Treasurer ..... Chas. Hunter Dunn, M.D.
Recorder and Editor ..... L. E. La Fétra, M.D.
Assistant Editor ..... O. M. Schloss, M.D.

COUNCIL

R. G. Freeman, M.D., *Chairman*

Alfred Hand, Jr., M.D.
L. E. La Fétra, M.D.
Isaac A. Abt, M.D.
John Howland, M.D.
Thomas Morgan Rotch, M.D.
D. M. Cowie, M.D.

MEETING PLACES

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.
1906. Atlantic City, N. J., May 30, 31 and June 1.

1907. Washington, May 7, 8 and 9.
1908. Delaware Water Gap, May 25, 26 and 27.
1909. Lenox, Mass., May 27 and 28.
1910. Washington, May 3, 4 and 5.
1911. Lake Mohonk, N. Y., May 31 and June 1 and 2.
1912. Hot Springs, Va., May 29, 30 and 31.
1913. Washington, May 5, 6 and 7.
1914. Stockbridge, Mass., May 26, 27 and 28.
1915. Lakewood, N. J., May 24, 25 and 26.

OFFICERS, 1915

President ..... George N. Acker, M.D.
Vice-President ..... Henry L. Coit, M.D.
Secretary ..... Samuel S. Adams, M.D.
Treasurer ..... Chas. Hunter Dunn, M.D.
Recorder and Editor ..... L. E. L. Fétra, M.D.
Assistant Editor ..... O. M. Schloss, M.D.

COUNCIL

Alfred Hand, Jr., M.D., *Chairman*

L. E. La Fétra, M.D. — Fritz B. Talbot, M.D.
Isaac A. Abt, M.D. — D. M. Cowie, M.D.
John Howland, M.D. — L. Emmett Holt, M.D.

MEMBERS

1903. Abt, Isaac A., M.D. ..... 4810 Kenwood Avenue, Chicago
1893. Acker, George N., M.D. ..... 913 Avenue of Presidents, Washington
O Adams, Samuel S., M.D. ..... 1801 Conecticutt Ave., N. W., Washington
1894. Baines, Allen M., M.D. ..... 228 Bloor Street, Toronto
O Blackader, A. D., M.D. ..... 236 Mountain Street, Montreal
1907. Bovaird, David, Jr., M.D. ..... 137 East Sixtieth Street, New York
1911. Bowditch, Henry I., M.D. ..... 416 Marlborough Street, Boston
1911. Butterworth, William W., M.D. .. 3914 Prytania Street, New Orleans, La.
O Caillé, Augustus, M.D. ..... 753 Madison Avenue, New York
1911. Carpenter, Howard C., M.D. ..... 1805 Spruce Street, Philadelphia
O Carr, Walter Lester, M.D. ..... 68 West Fifty-First Street, New York
O Chapin, Henry Dwight, M.D. .... 51 West Fifty First Street, New York
1897. Churchill, F. S., M.D. ..... 1259 North State Street, Chicago
1910. Coit, Henry L., M.D. ..... 277 Mt. Prospect Avenue, Newark, N.J.
1909. Cowie, D. M., M.D. ..... Lawrence Building, Ann Arbor, Mich.
1891. Crandall, Floyd M., M.D. ..... 113 West Ninety-Fifth Street, New York
O Dorning, John, M.D. ..... 124 West Eighty-First Street, New York
1906. Dunn, Charles Hunter, M.D. ..... 220 Marlborough Street, Boston
1904. Eaton, Percival J., M.D. .. 131 N. Highland Avenue, E. E., Pittsburgh
1907. Fife, Charles A., M.D. ..... 2033 Locust Street, Philadelphia
1895. Freeman, Rowland G., M.D. . 211 West Fifty-Seventh Street, New York
1913. Friedlander, Alfred, M.D. ..... 4 West Seventh Street, Cincinnati
1913. Gerstenberger, Henry J., M.D. ..... Osborn Building, Cleveland
1910. Gittings, J. C., M.D. ..... 3492 Chestnut Street, Philadelphia
1897. Graham, E. E., M.D. ..... 1713 Spruce Street, Philadelphia
1892. Griffith, J. P. Crozer, M.D. ..... 1810 Spruce Street, Philadelphia
1898. Hamill, S. McC., M.D. ..... 1822 Spruce Street, Philadelphia
1902. Hand, Alfred, Jr., M.D. ..... 1724 Pine Street, Philadelphia

1910. Haynes, Royal S., M.D.....213 West Seventieth Street, New York
1909. Heiman, Henry, M.D.....30 West Eighty-Eighth Street, New York
1914. Helmholz, Henry F., M.D.....1005 Michigan Avenue, Evanston, Ill.
1910. Herrman, Charles, M.D.....250 West Eighty-Eighth Street, New York
O Holt, L. Emmett, M.D.....14 West Fifty-Fifth Street, New York
1913. Hoobler, B. Raymond, M.D.....131 East Sixty-Ninth Street, New York
1905. Howland, John, M.D.....20 East Eager Street, Baltimore
O Huber, F., M.D.....209 East Seventeenth Street, New York
O Jacobi, A., M.D.....19 East Forty-Seventh Street, New York
1896. Kerley, Charles G., M.D.....132 West Eighty-First Street, New York
1905. Knox, J. H. Mason, M.D.....804 Cathedral Street, Baltimore
O Koplik, Henry, M.D.....30 East Sixty-Second Street, New York
1903. Ladd, Maynard, M.D.....270 Clarendon Street, Boston
1903. La Fétra, Linnæus E., M.D....113 East Sixty-First Street, New York
1911. Lucas, William Palmer, M.D...University of California, San Francisco
1912. McClanahan, H. M., M.D.....468 Brandeis Building, Omaha
1909. Machell, H. T., M.D.....95 Bellevue Avenue, W., Toronto
1907. Meara, Frank S., M.D.....400 West End Avenue, New York
1898. Miller, D. J. Milton, M.D....127 S. Illinois Avenue, Atlantic City, N. J.
1896. Morse, J. Lovett, M.D.....70 Bay State Road, Boston
1908. Nicoll, Matthias, Jr., M.D.....124 East Sixtieth Street, New York
O Northrup, William P., M.D...57 East Seventy-Ninth Street, New York
1910. Pisek, Godfrey R., M.D.....36 East Sixty-Second Street, New York
1912. Porter, R. Langley, M.D.....240 Stockton Street, San Francisco
1891. Rachford, B. K., M.D.....323 Broadway, Cincinnati
1905. Ruhräh, John, M.D.....839 North Eutaw Street, Baltimore
1900. Saunders, E. W., M.D.....3003 Lafayette Avenue, St. Louis, Mo.
1912. Schloss, Oscar M., M.D.....
.....54 West One Hundred and Fourth Street, New York
1913. Sedgwick, Julius P., M.D.....2015 Kenwood Parkway, Minneapolis
1902. Shaw, Henry L. K., M.D.....361 State Street, Albany
1914. Sherman, DeWitt H., M.D.....680 West Ferry Street, Buffalo
1891. Snow, Irving M., M.D.....476 Franklin Street, Buffalo
1905. Southworth, Thomas S., M.D.....807 Madison Avenue, New York
1911. Talbot, Fritz B., M.D.....311 Beacon Street, Boston
1910. Tileston, Wilder, M.D.....424 Temple Street, New Haven
1914. Van Ingen, Philip, M.D.....125 East Seventy-First Street, New York
1912. Veeder, Borden S., M.D.....1806 Locust Street, St. Louis, Mo.
1914. Walls, Frank X., M.D....1208-30 North Michigan Boulevard, Chicago
1895. Wentworth, A. H., M.D.....352 Marlborough Street, Boston
1896. Westcott, Thompson S., M.D.....1720 Pine Street, Philadelphia
1913. Wilcox, Herbert B., M.D.....159 East Seventieth Street, New York
O Winters, J. E., M.D.....25 West Thirty-Seventh Street, New York

Note.—The first column gives the date of election to the Society. The organizers of the Society are designated by "O," the date being 1888.

HONORARY MEMBERS

Dr. John Thompson.....Edinburgh, Scotland
Dr. George F. Still.....London, England
Dr. O. Heubner.....Berlin, Germany
Dr. William Osler.....Oxford, England
Dr. A. Baginsky.....Berlin, Germany
Dr. V. Hutinel.....Paris, France
Dr. Charles Rauchfuss.....St. Petersburg, Russia

Deceased

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

FREDERICK A. PACKARD, M.D.
Born, November 17, 1862,
Died, November 1, 1902.

WALTER S. CHRISTOPHER, M.D.
Born, 1859,
Died, March 2, 1905.

LEROY MILTON YALE, M.D.
Born, February 12, 1841,
Died, September 12, 1906.

JAMES PARK WEST, M.D.
Born, June 27, 1858,
Died, June 25, 1908.

FREDERICK FORCHHEIMER, M.D.
Born, 1853,
Died, June 1, 1913.

THOMAS MORGAN ROTCH, M.D.
Born, 1850,
Died, March 9, 1914.

CHARLES P. PUTNAM, M.D.
Born, 1845,
Died, April 22, 1914.

CONSTITUTION AND BY-LAWS OF THE AMERICAN PEDIATRIC SOCIETY

ARTICLE I

Name and Object of the Society

The Society shall be known as the American Pediatric Society, and shall hold an annual meeting.

It has for its object the advancement of the knowledge of physiology, pathology and therapeutics of infancy and childhood.

ARTICLE II

Proceedings

The proceedings shall consist of:

1. Discussions on subjects previously selected.
2. Original communications.
3. Demonstrations of gross and microscopic preparations, apparatus, and instruments.

ARTICLE III

Members

The Society shall be composed of three classes of members to be designated: (1) active members; (2) emeritus members, and (3) honorary members.

The number of active members shall be limited to seventy-five.

The number of emeritus members shall be unlimited.

The number of honorary members shall be limited to twenty-five.

ARTICLE IV

Election of Active Members

Nominations to membership, signed by two members of the Society, must be made in writing at least one meeting prior to election.

Nominations should be made to the Secretary, whose duty it shall be to require the nominators to write a personal letter endorsing and stating the qualifications of the nominee, and at the same time furnishing a list of the nominee's professional position and publications, with reprints of the latter, when obtainable.

It shall be the duty of the Secretary to transmit to the chairman of the council all the above papers pertaining to each nominee, at least three months prior to his possible election.*

* In the event that required information relating to candidates for membership is not furnished to the Secretary by three months before the time of the meeting at which they would be considered, such candidates for election shall be held over for another year. (Resolution adopted 1912.)

The chairman of the council shall transmit the above-named papers to the other members of the council in the order of their seniority as council members, the junior member of the council returning them to the Secretary.

It shall also be the duty of the Secretary to furnish a printed list of all nominees at least once a year to every member of the Society, and it shall be the duty of the members of the Society, without solicitation, to furnish the council with any information that they may possess as to the fitness of the nominees to become members of the Society.

On nomination by the council, members shall be elected by the Society by ballot. A two-thirds vote of the members present shall be necessary for election.

ARTICLE V

Election of Emeritus and Honorary Members

Active members who have served fifteen years and have paid all their dues, may on their request and on the recommendation of the Council be made emeritus members. They shall be entitled to attend meetings and read papers, when they so desire, but cannot vote.

Physicians of sufficient eminence to merit the distinction may be elected honorary members, to a number not exceeding twenty-five. Such members shall be entitled to attend all meetings and take part in the proceedings, but cannot vote. Honorary members shall be elected in the same way as active members.

ARTICLE VI

Initiation Fee and Annual Dues

Election to membership shall be completed by the payment of an initiation fee of $10.

Each active member shall pay an annual fee, the amount of which shall be decided at each annual meeting.

Emeritus and honorary members shall be exempt from fees.

ARTICLE VII

Officers

The officers shall consist of a President, Vice-President, Secretary, Recorder [and Editor], Treasurer and a Delegate to the Congress of Physicians and Surgeons.

The officers shall be elected annually on nomination by the council.

ARTICLE VIII

Duties of Officers

The duties of the President, Vice-President, Secretary and Treasurer shall be those usual to these offices. The Recorder [and Editor] shall secure the papers read and see that proper notes are taken of the discussion thereon for the use of the committee on publication.

ARTICLE IX

The Council

The council shall consist of seven members, the senior member being chairman. One member of the council shall be elected each year. Four members shall constitute a quorum. The senior member shall retire at the end of each year and shall not be immediately eligible to reelection.

ARTICLE X

Duties of Council

The duties of the council shall be to consider nominations for membership and report them to the Society. The votes of four members of the council shall be required for nomination. The council shall also nominate the officers of the Society and shall decide the time and place of meeting.

ARTICLE XI

Committee of Arrangements

The President, Secretary and the chairman of the council shall constitute a committee of arrangements, the President being chairman of this committee. They shall arrange the details of the meeting and the preparation of the program, and they shall have the authority to invite guests to attend the meeting and to participate in the discussion.

ARTICLE XII

Publication Committee

The Secretary, Treasurer and Recorder shall constitute a committee on publication, to which shall be referred all papers, reports and other matters intended for publication.

All papers presented shall become the property of the Society.

ARTICLE XIII

Amendment of Constitution and By-Laws

Proposals for amendments of the constitution and by-laws must have been made at the meeting previous to that at which they are

voted on, the notice for which shall contain an announcement of the proposed changes. Such changes shall require, for their adoption, an affirmative vote of three-fourths of the active members present.

ARTICLE XIV

Termination of Membership

A member may be expelled from the Society for conduct unbecoming a physician and a gentleman. In such cases, formal charges must be made in writing by two members, which shall be referred to the council.

Active membership shall lapse for any one of the following reasons:

(1) Absence from three consecutive meetings without excuse acceptable to the council; (2) failure to present and read a paper for five consecutive years; the Secretary in both these cases shall notify members one year before date of possible lapse; (3) non-payment of dues for two years, two notifications having been sent by the Treasurer.

ARTICLE XV

Quorum

Any number of members present at the appointed time of the annual meeting, shall constitute a quorum for the transaction of ordinary business, but for the election of members, fifteen shall be necessary for a quorum; and for the expulsion of members, or for altering the constitution and by-laws, twenty-five members shall be necessary.

ARTICLE XVI

Order of Business

1. The President shall call the meeting to order and deliver an annual address. In his absence the Vice-President shall preside, and in the absence of all these officers, the chairman of the council.

2. When a general discussion is arranged by the council, the two members appointed to open the discussion shall not occupy more than twenty minutes each; subsequent speakers shall be restricted to ten minutes each.

3. Papers shall not exceed twenty minutes in the reading. In the discussion following the reading of such papers, remarks shall be limited to ten minutes. Should any paper be too long to be read in

twenty minutes, the writer must prepare an abstract which can be read within that time.*

4. At the business session the report of the council as a committee on nominations to office and to membership, shall be made, and ballot shall be held thereon.

Adopted May 28, 1909.

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* In order to expedite the publication of the Transactions all members whose papers are to appear in the volume, be required either to have their articles in the hands of the editor before July 1 or to have them in process of publication in some medical journal by that date. In the latter event, the editor of the Transactions is to be notified what journal will publish the paper and reprints or galley proofs are to be furnished to him as soon as possible. (Resolution adopted 1912.)

MINUTES OF THE TWENTY-SIXTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY

Held at the Red Lion Inn, Stockbridge, Mass., May 26, 27 and 28, 1914

The meeting was called to order at 10:15 a. m. by the president, Dr. Samuel McC. Hamill, of Philadelphia. The following members were present during the sessions: Drs. Isaac A. Abt, Chicago; George N. Acker, Washington; Samuel S. Adams, Washington; A. D. Blackader, Montreal; Henry Ingersoll Bowditch, Boston; Walter Lester Carr, New York; Henry Dwight Chapin, New York; Frank S. Churchill, Chicago; Henry L. Coit, Newark; Floyd M. Crandall, New York; Charles Hunter Dunn, Boston; Percival J. Eaton, Pittsburgh; Charles A. Fife, Philadelphia; Rowland G. Freeman, New York; Alfred Friedlander, Cincinnati; Henry John Gerstenberger, Cleveland; J. P. Crozer Griffith, Philadelphia; S. McC. Hamill, Philadelphia; Henry Heiman, New York; Charles Herrman, New York; L. Emmett Holt, New York; A. Jacobi, New York; Charles G. Kerley, New York; J. H. M. Knox, Baltimore; L. E. La Fétra, New York; D. J. Milton Miller, Atlantic City; John Lovett Morse, Boston; William P. Northrup, New York; Godfrey R. Pisek, New York; B. K. Rachford, Cincinnati; John Ruhräh, Baltimore; Henry L. K. Shaw, Albany; Irving M. Snow, Buffalo; Thos. S. Southworth, New York; Fritz B. Talbot, Boston; Wilder Tileston, New Haven; B. S. Veeder, St. Louis; Herbert B. Wilcox, New York; J. E. Winters, New York.

There were introduced as guests: Drs. R. D. Moffett, New York; Philip Van Ingen, New York; Steelman, Kirk and Gordinnier, Troy; Smead, Toledo; Vanderpoel Adriance, Williamstown, Mass.; Colt, Pittsfield; Pennock, Pittsfield; Smith, Bridgeport; Stockwell, Stockbridge; Webster Miller, Stockbridge; Clifford S. Chapin, Great Barrington; W. W. Jones, Housatonic.

The minutes of the twenty-fifth annual meeting were adopted as published in the Transactions of the Society.

The following papers were read:

1. The President's Address by Dr. Samuel McC. Hamill.
2. Dr. John Ruhräh: "The Use of Dahlia."

The paper was discussed by Drs. Holt, Ruhräh, Kerley and Jacobi.

3. Dr. Alfred Friedlander: "Diagnosis of Whooping Cough by the Complement Deviation Test."

The paper was discussed by Drs. Talbot, La Fétra, Abt, Freeman, Kerley, Holt, and the discussion was closed by Dr. Friedlander.

4. Dr. Floyd M. Crandall: "Endocarditis in Children."

The paper was discussed by Drs. Chapin, Bowditch, Talbot, Griffith, Blackader, Coit, Abt, Tileston, Freeman, Miller, Eaton, Churchill, La Fétra, Hamill, Wilcox, and the discussion was closed by Dr. Crandall.

5. Dr. B. K. Rachford: "The Treatment of Recurrent Sibilant Bronchitis."

The paper was discussed by Drs. Eaton, Talbot, Pisek, Coit, Carr, Abt, Kerley, Adams, Miller, Griffith, Ruhräh, and the discussion was closed by Dr. Rachford.

At this point in the proceedings memorials of deceased members were read; that for Dr. Frederick Forchheimer by Dr. Rachford; the memorial of Dr. Charles Pickering Putnam by Dr. Morse and the memorial of Dr. Thomas Morgan Rotch by Dr. Jacobi.

6. Dr. Henry D. Chapin: "A Clinical Study of Typhoid Fever in Children."

The paper was discussed by Drs. Holt, Chapin, Adams, La Fétra and Jacobi.

7. Dr. Rowland G. Freeman: "Circumcision in the Masturbation of Female Infants."

The paper was discussed by Drs. Jacobi and Freeman.

8. Dr. John Lovett Morse: "Infantile Scorbutus and the Pasteurization of Milk."

The paper was discussed by Drs. Gerstenberger, Miller, Blackader, Northrup, Jacobi, Abt, Crandall, Freeman, Southworth, Pisek, Heiman, Griffith, Hamill, and the discussion was closed by Dr. Morse.

9. Dr. L. E. La Fétra: "Experience with High Calorie Diets in Typhoid Fever," by Drs. L. E. La Fétra and Louis C. Schroeder.

The paper was discussed by Drs. Chapin, Churchill, Friedlander, Kerley, Morse, and the discussion was closed by Dr. La Fétra.

10. Dr. J. C. Gittings: "The Caloric Value of the Diets of Sick Infants."

11. Drs. Fritz B. Talbot and Francis G. Benedict: "The Gaseous Metabolism of Infants."

The papers of Dr. Gittings and (conjointly) Drs. Talbot and Benedict were discussed by Drs. Dunn, Chapin, Veeder, Jacobi, Freeman, Kerley, and the discussion was closed by Drs. Gittings and Talbot.

12. Dr. D. J. Milton Miller: "The Feeding of Marasmic and Malnourished Infants on High Protein and Carbohydrates and Very Low Fat Values."

13. Dr. Percival J. Eaton: "Low Fats and High Proteids in Infant Feeding."

The papers of Dr. Miller and Dr. Eaton were discussed by Drs. Talbot, Morse, Jacobi, Chapin, Holt, Knox, Dunn, Abt, and the discussion was closed by Drs. Miller and Eaton.

14. Dr. L. Emmett Holt: "The Chemical Composition of Diarrheal Stools."

The paper was discussed by Drs. Southworth, Talbot, Kerley, and the discussion was closed by Dr. Holt.

15. Dr. Charles G. Kerley: "A Study of 141 Cases of Recurrent Vomiting in Private Practice."

The paper was discussed by Drs. Snow, Dunn, Friedlander, Abt, Griffith, Heiman, Ruhräh, Shaw, Knox, Northrup, and the discussion was closed by Dr. Kerley.

16. Dr. Rowland G. Freeman: "Weights and Measurements of Infants and Children."

The paper was discussed by Drs. Southworth, Pisek, Shaw, and the discussion was closed by Dr. Freeman.

17. Dr. J. P. Crozer Griffith: "Diseases Connected with Meckel's Diverticulum, with Special Reference to Diverticulitis."

The paper was discussed by Drs. Northrup, Blackader, and the discussion was closed by Dr. Griffith.

18. Dr. Isaac A. Abt: "Description of the New Sarah Morris Children's Hospital of Chicago" (with lantern-slide illustrations).

19. Dr. Henry L. Coit: "Anthropometric Methods, with Suggestions for Improvement, based on the Measurements of 540 Infants."

20. Dr. Henry Heiman: "The Artificial Cooling of Sick-Rooms in Summer—A Preliminary Report."

The paper was discussed by Drs. Northrup, Bowditch, Veeder, Adams, Shaw, and the discussion was closed by Dr. Heiman.

21. Dr. J. H. Mason Knox: "A Case of Primary Splenomegaly of the Gaucher Type."

22. Dr. Charles Herrman: "A Case of Gaucher's Disease—Splenectomy, with Recovery."

The papers of Dr. Knox and Dr. Herrman were discussed by Drs. Southworth, Holt, and the discussion was closed by Drs. Knox and Herrman.

23. Dr. Henry J. Gerstenberger: "Shall a Department of Pediatrics Include in Its Curriculum the Theoretical and Practical Training of Medical Students in the Social-Medical Work Amongst Infants and Children?"

The paper was discussed by Drs. Chapin, Blackader, Holt, Talbot, Kerley, Knox, Morse, Gittings, Hamill, and the discussion was closed by Dr. Gerstenberger.

24. Dr. Joseph E. Winters: "Pathogenesis and Prophylaxis of Rickets."

The paper was discussed by Drs. Freeman, Holt, Abt, La Fétra, Griffith, Talbot, and the discussion was closed by Dr. Winters.

25. Dr. E. A. Park: "Some Results of Thymus Extirpation," by Drs. John Howland, E. A. Park and R. D. McClure.

The paper was discussed by Drs. Friedlander, Abt, and the discussion was closed by Dr. Park.

26. Dr. Borden S. Veeder: "The Diagnosis and Treatment of the Late Type of Hereditary Syphilis," by Drs. Borden S. Veeder and P. C. Jeans.

The paper was discussed by Drs. Kerley and Hamill.

At this point in the proceedings Dr. Adams made a supplementary report of a case of erythromelalgia presented last year for diagnosis.

The following papers were read by title:

1. "Chylous Ascites in a Boy 8 Years of Age. Operation and surgical notes by Henry M. Silver, M.D.," by Dr. Francis Huber.

2. "A Case of Microcolon and Four Cases of Megalocolon," by Dr. Langley Porter.

3. "Abdominal Symptoms in Pleurisy and Pneumonia," by Dr. John Dorning.

4. "Some Observations on the Energy Metabolism of Normal and Atrophic Children, with Special Reference to the Specific Gravity of the Child's Body," by B. Raymond Hoobler, M.D., and J. R. Murlin, Ph.D.

5. "The Absorption of Unaltered Protein through the Intestinal Tract in Infants and Children," by Dr. Oscar M. Schloss.

6. "A Respiration Incubator for the Study of Metabolism in Infants," by J. R. Murlin, Ph.D. (by invitation).

7. "Primary Pulmonary Actinomycosis," by Drs. Francis Huber and Samuel Berkowitz.

EXECUTIVE SESSION, MAY 27, 1914, at 8:30 P. M.

The report of the Council was read by the chairman, Dr. Rowland G. Freeman.

The Council recommended that the resignations of Drs. A. C. Cotton, of Chicago; Charles G. Jennings, of Detroit, and Harold Williams, of Boston, be accepted with regret.

As officers for the ensuing year, for President, Dr. George N. Acker; Vice-President, Dr. Henry L. Coit; Secretary, Dr. S. S. Adams; Treasurer, Dr. C. H. Dunn; Editor, Dr. L. E. La Fétra; Assistant Editor, Dr. O. M. Schloss; Members of the Council, Dr. Fritz B. Talbot, to fill vacancy caused by the death of Dr. Rotch; Dr. L. Emmett Holt, for full term.

As delegates to the Congress of American Physicians, Dr. Abraham Jacobi and Dr. J. P. Crozer Griffith, Alternate.

As the place of meeting for 1915 the Council recommended Cape May, N. J., and the dates of meeting May 24, 25, 26, 1915.

That the assessment of $10 be made for the annual dues.

That the Transactions of the Society be printed by the American Medical Association, and that each volume be furnished with an author and subject index.

The Council recommended for election to membership in the Society Dr. F. X. Walls, Chicago; Dr. Philip Van Ingen, New York; Dr. H. F. Helmholtz, Chicago; Dr. De Witt H. Sherman, Buffalo.

That the President appoint the Committees mentioned in his Address, namely:

1. A Committee to Study the Child Labor Problem: Dr. Philip Van Ingen, Chairman; Drs. Henry L. K. Shaw and Herbert B. Wilcox.

2. A Committee to Study the Vulvovaginitis Problem: Dr. Howard C. Carpenter, Chairman; Drs. Charles A. Fife and J. C. Gittings.

3. A Committee to Cooperate with the Federal Children's Bureau: Dr. Samuel McC. Hamill, Chairman; Drs. L. Emmett Holt and Henry Dwight Chapin.

It was recommended by Council:

To establish a class of Emeritus Members. This class to consist of members who have served as active members for not less than 15 years, who request their transfer; such transfer being also recommended by the Council. Emeritus members to have the privilege of attendance and of reading papers at the meetings, but to pay no dues, to have no vote and to have no obligation to attend meetings or read papers except as they desire.

To this end the Council recommends amending the Constitution as follows: Art. III, line 1, substitute the word 3 for 2. Line 2, after Active Members, insert words (2) "emeritus members"; and after the word "and" (3) to replace (2). After line 3: "the number of emeritus members shall be unlimited."

Art. III will then read:

The Society shall be composed of three classes of members, to be designated: (1) active members; (2) emeritus members, and (3) honorary members.

The number of Active Members shall be limited to seventy-five; the number of emeritus members shall be unlimited; and the number of honorary members shall be limited to twenty-five.

Art. V to be amended as follows:

In title, after word "of" insert words "Emeritus and."

Immediately after title insert words: "Active Members who have served fifteen years and have paid all their dues, may on their request and on the recommendation of the Council be made emeritus members. They shall be entitled to attend meetings and read papers, when they so desire, but cannot vote."

Art. VI, last line, insert "Emeritus and" before word "honorary," so as to read, "Emeritus and Honorary Members shall be exempt from fees."

Art. XIV, line 4, insert word "Active" so as to read, "Active membership shall lapse for any one of the following reasons."

(1) Absence from three consecutive meetings without excuse acceptable to the Council; (2) Failure to present and read a paper for five consecutive years. The Secretary in both these cases shall notify members one year before date of possible lapse; (3) Non-payment of dues for two years, two notifications having been sent by the Treasurer.

The recommendations of the Council were adopted by the Society.

SAMUEL S. ADAMS, SECRETARY,
LINNAEUS E. LA FÉTRA, RECORDER.

RESPONSIBILITY OF THE PEDIATRIST TOWARD THE PROBLEMS OF PUBLIC HEALTH

SAMUEL McCLINTOCK HAMILL, M.D.
PHILADELPHIA

PRESIDENT'S ADDRESS

This society is one of the most distinguished organizations devoted to the study of disease in infants and children in the entire world. Its published transactions, covering the years from 1888 to the present, contain the most valuable contributions to pediatrics that have been published in the English language. Since its foundation its roll has contained the names of all the foremost pediatrists of the American continent.

To be elected to the presidency of such a society is a great honor, and one which every pediatrist must naturally covet. It is with a very deep sense of appreciation, and a full recognition of my unworthiness that I gratefully accept the high office to which you have called me.

Fate is inexorable. So it happens that as the years come and go, some of us fail to respond to the call for the annual meeting. This year Forchheimer, Rotch and Putnam are not with us. A call more mandatory than the appeal of our society has summoned them. They have given up their earnest, effective work among us, and hereafter they shall mingle with us only in spirit and memory. These gentlemen have been our friends, and as friends we shall miss them, but what we shall miss much more is their stimulating influence in this society, and above all, their contributions to the subject of pediatrics and the betterment of the world.

You were all friends of each of these men, and you will understand, therefore, that it is with no disrespect to the memory of any of them that I pay special tribute to the memory of one. We all know the burden of sorrow that rested on Rotch these many years. We know of his beautiful, untiring devotion to his invalid wife, and how, notwithstanding this ever-present cloud, he was the same patient, cheerful, hard-working, contributing Rotch — manifesting the keenest interest *always,* in everything that pertained to the advancement of his pro-

fession and his specialty. His life should be a help and inspiration to all of us. In our hours of discouragement and disappointment, when the obstacles to progress are looming large, we should remember what he overcame — how he sorrowed and worked on.

My text will be found on the seventeenth page of the first volume of the Transactions of this society. It reads as follows: "Questions of Public Hygiene and Medicine are both professional and social. Thus, every physician is by destiny a 'political being' in the sense in which the ancients defined the term; viz., a citizen of the commonwealth, with many rights and great responsibilities."

I am quoting from Jacobi's first Epistle to the Pediatricians, written in the year 1888 on the occasion of the first scientific meeting of the American Pediatric Society. These remarks were made at the close of Dr. Jacobi's presidential address, following that portion of it in which he was trying to indicate to the newly organized society the importance of its participating in the solution of the public-health problems of the day. Indeed, I have been so much impressed by the wisdom and far-sightedness of that address, that I wish I might appropriate it in its entirety as my address to you to-day. Even at the risk of going beyond the bounds of license, I shall take the liberty of quoting from it at considerable length.

After discussing the value of combined effort in obtaining results, Jacobi presents the most admirable and intelligent argument for the existence of pediatrics as a specialty that I have ever read. After defining at length the close and interdependent relationship of pediatrics to general medicine, surgery, neurology and all of the other branches of medicine, he emphasizes the relationship of pediatrics to the most vital questions of public hygiene. It is in this connection that he makes the following remarks:

"The physician, and particularly he who makes pediatrics his special study, is a pedagogue by profession. The question of school-house buildings and school-room furniture, the structure of bench and table, the paper and type in the books, the number of school hours for the average child and the individual pupil, the number and length of recesses, the hours and duration of intervening meals, the alternation of mental and physical training, the age at which the average and the individual child should be first sent, have been too long decided by school boards consisting of coal merchants, carpenters, cheap printers

and under-taught or over-aged school mistresses. Not, however, by physicians. . . .

"If there is a country in the world with a great destiny and a grave responsibility, it is ours. Its self-assumed destiny is to raise humanitarian and social development to a higher plane by amalgamating, humanizing and civilizing the scum of all the inferior races and nationalities which are congregating under the folds of our flag. Unless the education and training of the young is carried on according to the principles of sound and scientific physical and mental hygiene, neither the aim of our political institutions will ever be reached nor the United States fulfil its true manifest destiny. That manifest destiny is not so much the political one of excluding Europeans from our continent . . . but of raising the standard of physical and mental health to possible perfection, and thereby contributing to the welfare and happiness of the people."

It is thus seen that twenty-six years ago Jacobi indicated in very definite terms what he thought to be the duty of this society and its individual members to the problems of the commonwealth.

Dr. Jacobi is not the only one of our number that has emphasized this duty, but as has been his wont, he was the pioneer. Not until fifteen years later did Caillé address the society on the subject "The Influence of American Pediatric Societies in Promoting the Welfare of American Children." In this address he says: "The time has arrived when the weight and authority of our society, in addition to being felt by our professional colleagues, should be made to extend to the millions of homes throughout the land. In other words, the American Pediatric Society should now be ready to assume broader obligations and to grapple with larger problems.

"To shape and promote the public understanding regarding sanitation of the child's environment and to supervise from a medical standpoint the education of American children should be one of the chief aims of the American Pediatric Society." He suggested for consideration such themes as "ventilation in homes, temporary homes, private schools, boarding schools, etc.; discussion and criticism of the Sanitary Code as interpreted by Health Boards; School Hygiene and School Inspection."

Stimulated by Caillé's example, Jennings in the following year addressed the Society on "The Medical Supervision of Schools and the Progress of School Hygiene." In closing his address he said: "It may

not be the province of this society to introduce reforms in school hygiene or other departments of national sanitation. The objects and methods of its organization preclude this. It may and should be ready, however, with its exceptional knowledge of child pathology, to unite with national educational bodies, boards of health and similar public organizations, in movements directed to the improvement in the physical development and well-being of the children of the nation."

In 1908 Kerley presented a very interesting address on "Public School Education," dealing with the subject in a purely personal manner and making no suggestion as to any part this society might play in the solution of this great problem. He did, however, point out very definitely the importance of the pediatrist to the problem.

At the meeting in Lenox in 1909, the lamented Rotch read a paper before the society on the subject "The Position and Work of the American Pediatric Society Toward Public Questions." This admirable paper and the discussion which it stimulated, are probably fresh in your minds. Rotch aimed in this communication to indicate the duty of the society in the solution of the many problems that relate to the welfare of the child, that have been disposed of heretofore exclusively by the reformer and the philanthropist, neither of whom has a knowledge of or gives consideration to the medical aspect of these problems. He said: "It would seem almost the duty of the American Pediatric Society to use the great, wise and efficient influence which it surely is well fitted to exert, in assisting in the work of safeguarding early life by assuming an entirely independent position in the various communities where individual influence is felt and respected. We should make ourselves felt in the light of an advisory body, which shall from time to time bring up among the other subjects for discussion at our yearly meetings, one by one, the many important questions connected with child life."

The value of an anatomic index as a means of determining the fitness of the child for school and the physical work of early life, which was the basis of Rotch's paper, is not pertinent to the question we are dealing with to-day. One may or may not endorse his work, but the part of his paper defining the duty of this society in relation to public questions is important. In the discussion of the paper, the general principle advocated by Rotch was endorsed by La Fétra, Adams and Kerley, while Abt, Holt, Morse and Carr dissented. La Fétra presented an admirable, carefully prepared and extremely suggestive

endorsement which shall be referred to later. In dissenting, Dr. Abt said: "It seems to me that it is our mission to stimulate and encourage scientific work to the very highest degree. It should be the farthest from our purpose to become entangled in political or educational questions." Dr. Holt said: "I should feel sorry to see a large part of the work of this society devoted to subjects of this kind, which, though of sociologic interest, are not so much along the line of work of most of us as other matters more strictly medical. I believe we can do our best work along lines of research." Dr. Morse endorsed what Dr. Abt and Dr. Holt had said, and Dr. Carr thought that as individuals in our several communities we should be active, but, as a society, our work "should be largely medical."

In 1910, Edsall, in his presidential address, discussed the question under consideration and strongly advocated this society acting as a center for the accumulation and dissemination of knowledge relating to subjects which are intimately associated with pediatrics, and are, at the same time, of great importance to the public at large — such subjects, for instance, as education, schools, industrial conditions among children, etc.

Finally, in 1912, Carr, evidently having had a change of heart since his discussion of Rotch's paper, said in his presidential address: "The American Pediatric Society cannot stand as the highest pediatric body in this country if it fails to recognize the many influences that are laboring, both on this continent and in Europe, to lower the mortality of infancy and childhood; nor can its opinions be made authoritative unless it acts officially to cooperate with other agencies that are attempting to bring mortality and morbidity under control."

My reference to Dr. Carr's change of heart is not said in criticism, but rather in commendation, as it shows his ability to recognize the evolution of the times.

I have quoted rather freely from those of our number who have expressed to you from time to time their opinions regarding what they believe to be the opportunities of this society in the solution of the innumerable problems affecting the welfare of the child.

I have searched our Transactions from beginning to end in order that I might report to you what the society has actually done in this line. I find this interesting record:

At the meeting in Hot Springs, Va., in May, 1895, the following resolution was adopted: "Resolved, that in the opinion of the society

the evidence thus far produced regarding the effects of diphtheria antitoxin justifies its further and extensive trial." On motion of Caillé, the secretary was authorized to publish this resolution in a half dozen of the leading medical journals of the country.

At the meeting in Washington, D. C., in May, 1900, the following resolution was adopted: "Resolved, to petition the Congress of the United States to provide adequate facilities for verifying weights, measures and clinical apparatus."

At the meeting in Washington, D. C., in 1913, the following resolution was adopted: "Resolved, that until such time as it has been established by recognized medical authorities that inoculation with a secret bacterial preparation is without ill effects, this society is strongly opposed to its employment, as a preventive measure, in infants and young children."

These three are the only formal actions on subjects relating to the general welfare of infants and children that are recorded in our Transactions, and to place all of them in that category requires a fairly liberal interpretation.

Let me now return to and repeat my text: "Questions of Public Hygiene and Medicine are both professional and social. Thus every physician is by destiny a 'political being' in the sense in which the ancients defined the term; viz., a citizen of the commonwealth with many rights and great responsibilities."

We are citizens of the commonwealth and we have great responsibilities. As citizens of the commonwealth, are we meeting our responsibilities?

Of the seventy physicians comprising the membership of this society, each has been chosen because of some commendable contribution to the specialty we represent. We include in our roll the names of all the noteworthy teachers of pediatrics in America. In short, and in all humility, we are the leading pediatric organization of the English speaking people. It naturally follows, that the opinion of this society on all problems relating to the welfare of the child should be the most authoritative and valuable opinion obtainable. In the light of these facts, have we the right to refuse consideration of these vital problems in preventive medicine that are being unwisely and irrationally disposed of by men and women who are often swayed by sentiment and who are without the knowledge of medicine, on which, in the final analysis, every problem relating to the child must rest? I am prepared to answer "no" to this question.

At this point I wish to disclaim any reflection whatsoever on the splendid scientific accomplishments of the American Pediatric Society. Its record is unique. Nor do I want for one moment to convey the impression that I believe that the American Pediatric Society should sacrifice one particle of its scientific endeavor along the lines of laboratory and clinical research. What I do believe, however, is that it should add to its program scientific research along the lines of preventive medicine as it applies to the welfare of the child.

What, after all, is preventive medicine? It is synonymous with public health. The maintenance of the public health means practically the maintenance of the health of the child. The great bulk of mortality and morbidity occurs during the period of childhood, and unquestionably, the preventable diseases, which have as their basis defective public-health measures and which are largely responsible for this mortality and morbidity, are the diseases of early life. In other words, all public-health problems have a pediatric basis, and in their solution the pediatrist should be a potent factor.

Why should he be? Why should not these problems continue to be the problems of the charitably and philanthropically inclined reformer and the sociologist? The answer is simple. Fundamentally, they are medical problems, and neither the philanthropist nor the sociologist is competent to cope with them. Their solution requires special medical knowledge which the pediatrist possesses in high degree. This society is especially equipped to study and advise regarding most of these problems, many of which are being disposed of to-day to the detriment of the child and of the commonwealth. And yet we decline to participate in the solution of these grave questions, and practically say to the public that need us — "some of us as individuals may be glad to help you, but as a society specially organized for the study of the child, and possessed of the most advanced knowledge regarding his problems — the very knowledge which you most need and cannot get elsewhere — we have no interest in the welfare of your children; our problems are purely scientific."

Of course there are innmerable problems affecting the welfare of the child in which our individual members are deeply interested, and in the solution of which our society should participate. Birth registration, for instance, which is so essential to the study and control of infant mortality, is surely a subject in which we should have a vital

interest. And yet, as a society, we have never offered the slightest encouragement to those who have been endeavoring to make birth registration in this country of some value.

As Dr. La Fétra pointed out in his discussion of Dr. Rotch's paper, the question of the regulation of the midwife is one very closely related to the mortality of early infancy. Should we not in some way contribute to the solution of the midwife problem?

Dr. La Fétra also referred to the present unsatisfactory condition of the day nursery. The entire status of this institution demands very careful investigation and interpretation. Many of the day nurseries are organized and conducted without even a modicum of medical advice; the sanitary condition and direction of most of them is hopeless, and the licensing and control of day nurseries is accomplished in but two or three cities in the entire country. And yet this lack of control makes the day nursery one of the potent factors in the dissemination of disease.

We know, as does, perhaps, no other group of individuals in the land, the death-dealing power of most of the institutions that care for the foundling, the destitute child and the orphan. Have we no interest in the sanitary improvement of the best and the elimination of the unfit, or in some even more satisfactory solution of the institution question?

The problems of school hygiene — have we no interest in them? Have the school curriculum, the school building, the medical inspection of schools, the fresh-air school, the athletic side of school life and the, at present, very much debated question of the feeding of schoolchildren no interest for us?

Should not the private schools of our cities and even our boarding schools look to us for guidance in the solution of their problems? Or, to put it differently, should not we express an opinion regarding the gross limitations of the majority of such institutions in relation to their effect on the mental and physical development of the child? Many of the private schools for boys and the great majority of the girls' schools, as housed in our cities, are a menace to the public health. They are receiving neither advice nor supervision. We pediatrists see the fruits of their deficiencies. Can we not at least protest?

I do not at all agree with the statement of Dr. Abt in his discussion of Dr. Rotch's paper, when he says: "When we deal with children who are employed in factories and stores, or if we concern ourselves with child labor laws, we are encroaching on the borderland. The internist

on the one hand, and the sociologist on the other, are already laying claim to the problems of this period of life." In the first place this statement misrepresents the aims of the child-labor organizations. What they are attempting to do is to regulate the employment of children under the age of 14 — the age period which of course belongs to the pediatrician. In the second place, the internist, unfortunately, is not taking any more interest in this problem than the pediatrist. And in the third place, even though he wanted to make it such, the problem is not one for the sociologist to determine alone. The sociologist needs, and as a matter of fact he wants, the assistance of the pediatrist. In the early part of last year, when some child-labor legislation was pending in Pennsylvania, I was asked by a sociologist whether this very society had ever taken any official action or made any recommendations in relation to child-labor legislation.

The medical profession owes a great debt to the sociologist. He has revolutionized hospital practice by teaching us that the social study of the hospital patient is absolutely essential to the successful diagnosis and treatment of his case. On the other hand, without the pediatrist, the work of the sociologist, as it relates to the child, is of little value, and may even be dangerous. In other words, the problems of the physician and the sociologist are inseparable.

That is well illustrated in the very matter of the health aspects of the child labor problem. As Dr. Rotch has well pointed out, the knowledge which the pediatrist alone can give will prevent the enactment of laws which are unwise and unjust to both the child and his parents and employer.

In recent years, the health departments of some of our cities have made special provision for the care of the city child, by the creation of departments of child hygiene. Should not this society do everything in its power to stimulate the creation of such departments and outline their scope and limitations? It is through the medium of such departments, rightly conducted, that the terrific morbidity and mortality among infants and children will be reduced. New York's remarkable record is a splendid illustration of their possibilities. And yet we have never contributed anything to the development of this promising work.

Very recently an attempt was made to cripple seriously the work of the National Children's Bureau, established as a result of the child labor movement. Would it not have been of immense value to the chief of that bureau to have had the endorsement of the American

Pediatric Society, and to have had it recorded in the minutes of this society that it had appointed a committee from its membership which stood ready to act in an advisory capacity to the chief of the bureau whenever she cared to call on it? It is vital to the welfare of the American child that this bureau be developed to the highest degree of efficiency. What has this society done to help it attain this end?

There are innumerable problems, gentlemen, that could be put up to you for consideration. Think, for instance, of the programs of the charitable, philanthropic, educational and social institutions in our cities, most of which are in part public-health programs, and most of which also are formulated and carried out without medical advice. How is this unfortunate condition to be relieved? Perhaps as individuals in our various communities we can do and are doing something. The pediatrist, however, has withheld his assistance and influence so long that the lay organizations have learned to do without him. They have lost sight of the medical aspects of their problems, or they have assumed that they possess the necessary ability to solve them. This attitude has become so fixed that it requires more than the influence of the individual pediatrist to change it. What is needed is the carefully formulated opinion of the foremost pediatric society. The guidance and opinion of such an organization they must and will respect. Those of us who have been in close touch with these organizations understand the crying need of the assistance that such a society as this can render.

As has been pointed out in the earlier part of this communication, this question of the welfare of the child has been presented for your consideration on a number of occasions, and it seems to me that it is quite time for us to render an answer to the question: Is it or is it not the duty of the American Pediatric Society to take an authoritative position in relation to the important sociologic or public-health problems which affect the welfare of the child?

As I have already said, I believe it to be our duty. If not, on what organization shall this duty rest? There is no other equipped as we are.

The disposition of these grave problems is at present in the hands of the laity. Sentiment, a lack of knowledge, and bad judgment are leading to the establishment of laws, regulations and customs that will eventually menace, rather than benefit the human race. This society, by reason of its peculiar fitness, has an opportunity to render a service to humanity that is given to few other organizations.

I wish to repeat what I have already said, that in taking up a study of the problems that have been under discussion, I do not believe that the American Pediatric Society should sacrifice one particle of its scientific endeavor along the lines of laboratory and clinical research. I can say with Dr. Holt that "I should feel sorry to see a large part of the work of this society devoted to subjects of this kind"; but they are subjects which have much more than a sociologic interest — they are problems of profound medical interest and form the basis of the maintenance of the health of the child. The maintenance of the health of the child, in turn, is the foundation on which the future of the human race must be built. Much of the invalidism of the adult, mental and physical, much of the moral obliquity of the child and the adult, have as their foundation the neglect of the medical aspect of these sociologic problems. The importance of the medical man, and especially the pediatrist, to these questions, is being more fully recognized each day, and in the light of these facts, I do not see how it is possible for this peculiarily qualified organization to continue longer its attitude of indifference. We have already lost a great opportunity in not earlier appreciating the wisdom of Jacobi's advice. Let us not continue to neglect it.

As to the method of procedure, I think the suggestion of Edsall that this society act as a center for the accumulation and dissemination of knowledge relating to these subjects, is admirable. We cannot treat all of the innumerable themes that are awaiting us at once. As Rotch has suggested, "we must bring these subjects up for discussion, one by one, at our yearly meetings."

I would therefore recommend that at this meeting we appoint three committees, consisting of three members each — the one to study thoroughly the present status of the child labor problem, and to present a critical and constructive report at the next annual meeting; the second to study the vulvovaginitis problem in its relationship to the public health, its report to be submitted at the same meeting, and the third to be known as the Committee on Co-operation with the Children's Bureau of the United States Department of Labor.

1822 Spruce Street.

THE USE OF DAHLIA IN INFECTIONS

JOHN RUHRÄH, M.D.
BALTIMORE

For a long time anilin dyes have been known to exert certain antiseptic actions and this has been the subject of a considerable amount of study and certain practical applications have been made as the result. As early as 1886, Pfeffer showed the effects of certain anilin dyes on the cells of the higher plants and in the following year Rozsahegyi pointed out the harmful action on certain bacteria. He also observed the selective action of the dyes. For example, the bacillus of rabbit septicemia grows readily in the presence of carmine and vesuvin while it will not grow in the presence of gentian violet. The bacillus of chicken cholera does not grow in the presence of vesuvin but will grow in the presence of gentian violet. These observations did not attract much attention but a few years later, in 1890, a number of communications were made dealing with this subject. Stilling showed that the dye not only exerts an antiseptic action but also actually kills certain bacteria and at his request the firm of Merck and Company put out a mixture of methyl violet 6B, dahlia and benzoyl violet. This was called blue pyoktanin. Stilling mentions the fact that Kramenski suggested the use of pure anilin and of anilin dyes as inhalations in the treatment of tuberculosis of the lungs and it seems that the curative action of anilin on wounds has been known to workers in the dye factories where it is the custom to treat skin wounds by dusting them with various dyes. The selective action of these dyes led to their use in culture media and Drigalski and Conradi used crystal violet in a dilution of 1 to 100,000 in a culture medium designed for the isolation of the typhoid bacillus. They believed that it hindered the growth of various cocci without interfering with the growth of the typhoid bacillus. Churchman[1] recently published the details of a study of the effect of gentian violet on various bacteria and found out that the various forms of organisms could be separated into violet positive or those in which the growth is inhibited and violet negative in which the growth is not inhibited.

1. Churchman: Jour. Exper. Med., 1912, xvi, 221 and 822; Ibid 1913, xvii, 373.

For almost all bacteria this relation of the growth to gentian violet is quite constant. Simon and Wood[2] have made a study and have established the fact that an acid dye irrespective of its color in the standard concentration of from 1 to 100,000 is devoid of bactericidal properties while a basic dye likewise irrespective of its color possesses inhibitory power. Among the basic dyes may be mentioned basic fuchsin, methyl violet, crystal violet, Hoffman's violet and dahlia; among the acid dyes, the acid fuchsin, both violet and acid violet. Simon states that this inhibitory effect on the growth of bacteria is not referable to the color but to their chemical structure (triamino-triphenyl methanes). A second contribution by Simon and Wood[3] contains an account of numerous experiments with various dyes in which they show that the inhibitory action on the growth of certain bacteria which has been demonstrated to be common to all the tri-aminotriphenyl methanes is not an exclusive property of this group of anilin dyes but is manifested also to a greater or less extent by other strongly basic dyes. A theory is given concerning this which need not be gone into at this time. They have also demonstrated that certain bacteria belonging to groups ordinarily susceptible to dyes may grow in their presence and many organisms may overcome the susceptibility by adaptation. This has also been announced from Ehrlich's laboratory. Similar studies have been made by May[4] and by May and Heidingsfeld.[5] They found that in studying the fuchsins, essentially the same thing that is the most basic, rosalin acetate, is also most toxic for bacteria while the acid fuchsins are not toxic at all. They found that a 1 to 1,000 solution of basic fuchsin would kill in five minutes typhoid, paratyphoid, tubercle bacillus, staphylococci and the *Oidium albicans*. They also found that the 1 per cent. solution did not produce any irritation of the mucous membranes and could even be injected into the bladder. They also found that 1 per cent. ointments which could be used in the treatment of ulcers and abrasions were non-irritating. Stronger ointments would occasionally produce some irritation. They suggest the base of the ointment be made of five parts of petrolatum and five parts lanolin.

2. Simon and Wood: Am. Jour. Med. Sc., February, 1914, p. 274.
3. Simon and Wood: Am. Jour. Med. Sc., April, 1914, p. 524.
4. May: Jour. Am. Med. Assn., April 20, 1912, p. 1174.
5. May and Heidingsfeld: Jour. Am. Med., Assn., May 31, 1913, p. 1680.

About two years ago in searching for an efficient local application for streptococcus infections of the throat, Dr. Charles Simon suggested the use of dahlia. I started this as a local application, beginning at first with very weak solutions, and I soon found that the saturated, that is about 4 per cent., solution could be applied to the mucous membranes of the throat or, in fact, other parts of the body without producing either pain or subsequent irritation. The drug seems to penetrate only to short distances and for the deeper seated affections has no value; but for superficial involvement of the mucous membranes, whether the infection is due to streptococcus or to other organisms the effect is quite striking. In some cases very little effect is noted, it is true, but in others there is marked lessening of the intensity of the inflammation and coincidently a marked lessening of the constitutional symptoms. It has the advantage over other applications in that it is not painful, does not produce irritation and is markedly antiseptic. The only disadvantage is the color, which of course will stain fabrics with which it comes in contact, although most of these stains can be removed if the garment is immediately washed out in cold water.

For ulcerations about the mouth it may be used either by applying a saturated solution or a mouth wash varying in strength from 1 to 1,000 or 1 to 10,000 may be used. The stronger solutions need not be used very frequently. The dahlia not only kills the offending organism but it has a marked stimulating effect on the healing. Externally on skin surfaces the drug may be used with very marked benefit particularly on ulcerations. I have used it with remarkable benefit on vaccinations which were slow in healing and on other abraded surfaces particularly those which are infected. I have not had an opportunity of using it in erysipelas but Dr. Louis P. Hamburger of Baltimore, and Dr. T. B. Johnson of Frederick, have used it in a number of cases with the most satisfactory results. Dr. Johnson informs me that he has used it with remarkable success in both acute and chronic eczema, in herpes, tinea tonsurans and furunculosis. In one resistant case of tinea sycosis in which the whole surface of the beard was tremendously involved, the patient was cured after five or six daily applications. It may be used with reasonable hope of success in skin lesions caused by or accompanied with pus organisms.

DISCUSSION

DR. HOLT: I would like to ask Dr. Ruhräh whether he has used dahlia in diphtheria cases.

DR. RUHRÄH: I have used it in a few, but not in a sufficient number of cases. In the few instances in which I have used it, it has worked very well. It has to be applied very thoroughly with a dropper through the nose. You can use a strong solution if you make one application; or several applications may be made of the weaker solutions.

DR. KERLEY: Would it be of any use in ivy poisoning?

DR. RUHRÄH: I have not tried it. I don't know.

DR. KERLEY: What do you think about it?

DR. RUHRÄH: It would not do any harm to try it.

DR. JACOBI: I only wish to take a single minute for the purpose of referring to what I published some eight years ago on the effect of methylene blue. In cases of chronic cancer you can do a great deal by giving methylene blue, not subcutaneously, as Mosetig-Moorhof proposed to twenty-four years ago, but internally. The paper was published in the *Journal of the American Medical Association,* and has been overlooked and neglected in the most conscientious manner, as many good things are thus forgotten. I have used methylene blue in that way now, internally, for the last twenty-three years. Those who are interested in it, will find my short paper on indications, doses and method of administration in the *Journal of the American Medical Association* of Nov. 10, 1906; it is called "Methylthionin Hydrochlorid," which is its chemical name.

DIAGNOSIS OF WHOOPING-COUGH BY THE COMPLEMENT-DEVIATION TEST

ALFRED FRIEDLANDER, M.D. AND E. A. WAGNER, M.D.
CINCINNATI

The recognition of such a scourge as whooping-cough, particularly when occurring in institutions in which children are segregated in large numbers, is a matter of great importance.

In his address on this subject before this Society last year, Morse emphasized the difficulty—and the value—of early diagnosis.

By the time the paroxysmal stage is reached, the infection, where children are gathered together, is widespread, and the resulting mortality high, as shown by carefully collated statistics.

The desire to find a definite method of early diagnosis led us to a study of the complement-deviation test. With our modified technic we have been able to make the diagnosis of pertussis in all stages of the disease, catarrhal, paroxysmal and convalescent. Particular emphasis is laid on the point that the diagnosis can be made early in the catarrhal stage, long before any whoop appears, and at a time when prompt isolation of the infected child will prevent the spread of the disease. Thus several children admitted to the open children's ward of the Cincinnati Hospital with a diagnosis of bronchitis, gave a positive early reaction, though there was neither vomiting nor whoop. They were immediately removed to the pertussis ward, and our open ward remained free of pertussis all winter. A preliminary report of our work has already been published[1] from which the following excerpt is taken.

When Bordet and Gengou[2] described the bacillus of whooping-cough in 1906 they used the complement-deviation test to control their bacteriological findings. From this time on, it has been known that the blood of patients in the late stages of pertussis, during convalescence and for some time thereafter, would give a positive test showing definite deviation of complement. More recently it has been shown that

1. Friedlander, A., and Wagner, E. A.: Jour. Am. Med. Assn., March 28, 1914, p. 1008.
2. Bordet and Gengou: Ann. de l'Inst. Pasteur, 1906, xx, 731.

in the later stages of atypical cases of pertussis, it is possible to determine the specific pertussis character of the infection by means of this test.[3]

Several observers have denied the possibility of making a diagnosis of pertussis even during the height of a typical attack or directly after convalescence by means of the complement-deviation test. Working in the Royal Serological Institute of Vienna, Bächer and Menschikoff[4] report that in twenty-seven cases of pertussis, moderate and severe, in the height of the attack, and in convalescence, attempts were made to obtain positive complement-deviation reactions, without success in a single case. Only after vaccines, prepared from pure cultures of the Bordet-Gengou bacillus were given was the test ever positive.

TABLE 1.—RESULTS OF COMPLEMENT-DEVIATION TESTS FOR WHOOPING-COUGH

	No.	Positive	Negative	Per Cent.
Cases whooping	18	18	0	100
Normals	16	0	16	100
Early cases catarrhal stage	12	11	1	91.6
Not whooping; course otherwise typical	1	1	0	100

Commenting on these and other similar findings, Bordet[5] himself says: "I repeat that the power (of fixing the complement) is not seen early. In general it does not show itself markedly till toward the period of convalescence or cure." Netter and Weil[6] have reached practically the same conclusion. In fact it would appear to be the consensus of opinion that the test has no diagnostic value in the early stages.

Our own results lead us to the opinion that the complement-deviation test is of the greatest possible value in the diagnosis at all stages. Our results are herewith presented in tabular form.

3. Bordet and Gengou: Centralbl. f. Bakteriol., Abt. I, Orig., 1911, lviii, 537; Bordet and Brunard: Bull. Acad. de méd. Belge, 1910, xxiv, 320; Delcourt: Presse méd. Belge, 1912, lxiv, 19.

4. Bächer and Menschikoff: Centralbl. f. Bakteriol., Abt. I, Orig., 1912, lxi, 218.

5. Bordet: Centralbl. f. Bakteriol., Abt. I, Orig., 1912, lxvi, 276.

6. Netter and Weil: Compt. rend. Soc. de biol., 1913, lxxiv, 236.

These cases were taken both from our hospital service and from private practice, and all cases were carefully followed.

It will be noted that the 18 cases tested during the paroxysmal stage gave positive reactions: 3 + in each case. Nine of the cases were in the first week of the whoop, and 3 early in the second week. In no case did a normal give a positive reaction. This series included several cases of bronchitis. Of the 12 early cases, tested in the catarrhal stage, 11 gave positive reactions, and in each instance the definite whoop appeared later, thus confirming the serologic diagnosis clinically. The one case that gave a negative reaction was tested at the very beginning of the cough. The sister had given a positive test. Two weeks later the child with the negative test began to whoop.

TABLE 2.—TABLE OF ELEVEN POSITIVE REACTIONS TESTED IN THE CATARRHAL STAGE

No.	Duration of Cough Without Whoop	Time of Appearance of Whoop After Test
1	3 weeks	1 week
2	3 weeks	3 weeks
3	3 days	2 weeks
4	8 days	About 1 week
5	5 days	2 days
6	1 week	2 days
7	1 week	2 days
8	10 days	2 weeks
9	5 days	2 days
10	10 days	1 week
11	2 weeks	5 days

The one case giving a positive test that never whooped, occurred in a child with paroxysmal cough and vomiting lasting over a period of four weeks, evidently an atypical pertussis.

TECHNIC OF TEST

Our technic for the test has been as follows: A small amount of blood—about 15 to 20 drops—was taken from the patient's ear, finger or toe in small test-tubes or the Wright capillary tubes. For young children we have found the great toe very satisfactory.

The blood was kept at room temperature or placed in the incubator until coagulation had taken place. Serum was then separated more completely from the clot in the centrifuge. So far in our test we have used only fresh, active serum. Two drops of the serum were used in each test.

Hemolytic System: The Noguchi system was used because of its extreme delicacy and because of the small amounts of material — especially serum — required. In this system washed human corpuscles, 1 drop to 4 c.c. of salt water, are used.

Amboceptor: The amboceptor employed was prepared according to the Noguchi method, the serum being dried on filter paper. The complement was obtained in the usual way from guinea-pig — dilution 1 to 40. Aside from the delicacy of this hemolytic system, it is of great value in working with children because of the very small quantity of blood required. It is not necessary to take blood from the veins and the small quantity of blood required is easily obtained even from very young children.

Antigen: This is the most important factor in the test. The Bordet-Gengou bacillus was obtained in pure culture from the laboratories of Parke, Davis & Co. Most of our work was carried on with this culture. Cultures were also obtained from the H. K. Mulford Co., and from Dr. F. B. Mallory of Boston. The latter culture came from a strain grown at Theobald Smith's laboratory.

Subcultures were made on Bordet's medium and an ascitic fluid agar. Latterly we have used the ascitic fluid agar exclusively and our antigens were made up as follows: Seventy-two-hour growths were taken. The colonies, which were very tenacious, were scraped off the agar with a glass hook into sterile salt water. An emulsion was made and the bacteria again washed in salt water. It is important to do the second washing so as to rid the emulsion of any particles of agar. From this washed emulsion a standard suspension was made, and 0.1 to 0.2 c.c. of this used in the tests. Throughout the test, live bacteria were used. It will be noted that we have always used fresh, active serum and live bacteria, and we consider this innovation of great importance.

Controls: In each test known normal and known positive controls were used. In each series of tests the hemolytic system was tried out in the usual manner, using a water bath at 37 C. for incubation. After primary incubation for half an hour, the amount of amboceptor indicated by the preliminary test was added to our final test-tubes and the tubes again incubated in the water-bath.

In every instance independent readings were taken by each of us, always without previous knowledge of the clinical history of the given case.

The test is not at all difficult to carry out for any one with experience in serologic work. Using the precautions above noted, the readings have always been sharp, clear and unmistakable. It is not difficult to keep the bacillus growing on the ascitic fluid agar, which for the purposes of the test is a much more satisfactory medium than the original Bordet.

The point of particular importance is that by this method and by this test the diagnosis of pertussis can be made with absolute certainty in the catarrhal stage. Finally it is a matter of record that the success

of vaccine therapy depends in large measure on the time of its application. The earlier the vaccine is given the better the results. If it be possible to diagnosticate whooping-cough in the catarrhal stage surely and definitely, its rapid cure seems assured. We have just recently begun therapeutic vaccine work. In two cases—father and son—occurring in the private practice of one of us, the test was positive in the catarrhal stage in both instances. Both patients whooped subsequently, confirming the diagnosis clinically. Both of these cases were treated with a vaccine prepared from one of our own subcultures. The father received five injections at four-day intervals, 600,000,000 bacteria being given at a dose. In three weeks his cough had ceased completely. The boy also received five injections of same dosage, and in three and a half weeks his cough was over.

We believe, therefore, that the test as outlined is worthy of full confidence and that it offers definite early diagnosis and thus points the way to a rapid cure of pertussis.

4 West Seventh St.—3104 Jefferson Avenue.

DISCUSSION

DR. TALBOT: How early in the catarrhal stage does the test become positive?

DR. LAFÉTRA: I recall having read, about two years ago, in the *Archives de Médécin des Enfants,* the experience of a physician in charge of an infant asylum, who found that by using this test he could separate his patients so that he could prevent the spread of epidemics in the asylum; and though at that time I knew of Bordet's statement that the test was not found specific early in the disease, it seemed to me convincing from the character of this work that it offered a valuable means of controlling whooping-cough in institutions.

DR. ABT: I should like to ask the essayist why he doesn't mention the early bacteriologic diagnosis and what the possibilities are of early bacteriologic diagnosis?

DR. FREEMAN: While this work of Dr. Friedlander's is interesting and seems promising I have understood that other observers following the same method have obtained negative results. I suppose it is still uncertain whether we know the specific organism of whooping-cough.

DR. KERLEY: I had occasion, or rather opportunity, last summer to see considerable of the work of Wright and Freeman at St. Mary's Hospital, London; Dr. Freeman had charge of the experimental work in the use of pertussis vaccines. He had used it with 1,134 cases, and in each case he established a control of another child; his results, or at least his opinion as regards results was rather vague. He did not know whether or not the vaccines were of much value. I pressed him for his opinion; and he said he thought probably they did some good. He gave injections of 200,000,000 bacilli twice a week. In a certain series of cases it would seem that the injections were a benefit; and in others the salt solution which he used with the control appeared to be as

effective. He was not enthusiastic regarding the use of vaccines. He felt that when he used the pertussis vaccines in combination with the pneumococcus the results were somewhat better.

DR. HOLT: The close correspondence in clinical symptoms between certain cases of influenza and whooping-cough make it seem to me extremely desirable that these two diseases should be very carefully differentiated bacteriologically. During the past winter and spring we have had a great deal of influenza in the Babies' Hospital, so much so that we went through the institution at one time and took sputum cultures from every inmate of the house. At that time the resident physician had an attack which was clinically whooping-cough. He had, many times a day, severe paroxysms of coughing, which two or three times a day were attended by vomiting. The attack lasted about six weeks. His sputum showed no Bordet's bacilli but, repeatedly, great numbers of influenza bacilli, almost in pure culture. We did not have a case of whooping-cough in the hospital among the patients admitted, nor did one develop in the wards during the season. Such cases as this might explain some of the negative results obtained in Dr. Friedlander's work.

DR. FRIEDLANDER: With reference to Dr. Talbot's question as to time of cough in the early cases, I will say that Patient 3 had coughed for three days; Patient 4, eight days; Patient 5, four days. Two of the patients had been coughing for three weeks, one for one week and one for ten days.

With reference to the histories of the cases, I may say that they were very carefully taken. All details were obtained with accuracy. Many of the cases occurred in the children's ward of the city hospital, so that these children could be very carefully watched. The actual time of onset of cough could be ascertained without difficulty.

It is certainly true that in several instances children had a pertussoid cough. The diagnosis of influenza had been made, and all of these patients gave negative tests. With reference to the specificity of the organism it may be pointed out that we never got a positive reaction in a case that did not turn out clinically to be whooping-cough. In some of our adult patients who had had whooping-cough some time before, we got positive tests. In one case when the infection had occurred a year previously, and in another eighteen months. So far as the use of vaccines is concerned, there is of course still much doubt as to their nature. But I should like to point out that the stock vaccines as commercially sold contain relatively small doses — 50,000,000. In our therapeutic work with our own vaccines we have used much larger dosage, beginning with 125,000,000 and running up to 300,000,000. We have had no bad results from the vaccines and the therapeutic value seems to be distinct.

ENDOCARDITIS IN CHILDREN

BY FLOYD M. CRANDALL, M.D.
NEW YORK

It is not my purpose in this paper to present a systematic monograph on heart disease in children, but rather to record certain views reached after many years of observation. My first paper before this Society was a study of 142 cases of heart disease in children and was presented in 1891. It was based upon cases recorded by Dr. Holt at the Northwestern dispensary and others seen by me in his service at the New York Polyclinic. Since that time I have taken much interest in diseases of the rheumatic series, and hope that some of my experience is worthy of record. Last year Dr. Dunn read before this Society an admirable statistical paper based on 300 cases of heart disease seen in hospital practice. In reading that paper it seemed to me that one on the same subject from a different standpoint might be of some value. I am, therefore, presenting certain opinions, based not on statistics, but on personal experience. It is proper to say that these conclusions are not based on impressions and memory alone, which form a very uncertain basis for medical opinions, but upon a study of case histories of private patients, some of whom have been followed for many years. My general conclusion is that cardiac disease in private practice runs a considerably more favorable course than it does in hospital and dispensary practice. The reasons for this are obvious.

In selecting the title for this paper, I hesitated between using the term "carditis" or "endocarditis," but decided upon the latter. I am well aware that in the graver forms of heart disease which come to autopsy the disease is never limited to the endocardium alone. The pericardium and the myocardium are also involved. This was the experience of Sturges twenty or more years ago, and has been the constant experience of pathologists since that time. It is undoubtedly true that a certain amount of degeneration of the fibers of the heart muscle occurs during the course of every acute febrile disease or intoxication. This is frequently mild in degree, but myocarditis with little or no valvular involvement may cause the death of the patient, as in diphtheria. In watching the course of these cases, it is difficult to believe that in some of the less severe types, which run a favorable

course, any marked degree of myocarditis or pericarditis is present. It certainly seems that a poison like that of rheumatism circulating in the blood may so irritate the sentitive myocardium as to cause the primary and essential lesion in that membrane, rendering the designation of endocarditis proper. The probability of grave myocarditis, however, must never be forgotten. Examinations for its detection should be frequent and careful, for upon it, more than any other element, depends the prognosis and much of the treatment.

I have come to rely upon four symptoms as particularly suggestive of muscle involvement. The earliest to appear is irregular heart action or palpitation, and this is usually the most distinctive symptom. Syncope is characteristic of decided myocarditis, particularly in diphtheria. In addition to these two symptoms, cyanosis and precordial distress are very suggestive of muscle involvement. One or more of these symptoms in conjunction with physical signs warrants us in belief that in addition to the endocardial disease we have also muscle involvement. In the later stages the occurrence of cyanosis and edema is of more grave significance in the child than in the adult. They indicate a grave type of carditis, certainly endocardial and myocardial, and perhaps pericardial as well. It is usually impossible in a young child to determine the condition of the heart muscle, unless an examination can be made during sleep as well as during waking hours.

In determining the condition of the heart muscle, a blood examination may give assistance in addition to the aid usually expected of it. Palpitation and shortness of breath are such frequent accompaniments of anemia that it is helpful to ascertain the blood condition, and not always jump to the conclusion that the symptoms are wholly due to myocarditis. If the anemia is found to be marked, the prognosis may not be so gloomy, for its relief may directly mitigate the symptoms.

A point of great interest to me has been the occasional rapidity of the development of the physical signs of endocarditis. I have heard a loud endocardial murmur fully developed in a closely watched case within eighteen hours. It is not always the fact that a soft indefinite murmur precedes the development of distinctive sounds. Recognition of this fact would sometimes have saved criticism of practitioners by others who have been brought into the case and I think should be remembered before strictures are passed. It is quite possible that a distinctive blowing murmur may be present today that was not present yesterday. It is possible that the advent of fever with increased heart's

action may sometimes aid in the apparent rapid development of endocardial lesions. It is a common statement in text-books that the severity of the case is not in proportion to the amount of sound heard on auscultation. There is truth in this statement if the case is judged by the amount of sound alone. If the murmurs are judged as they should be in connection with other signs, then the statement is not wholly true. Other things being equal, I find it hard to feel, when studying a case, that a loud rasping murmur is not more serious than a soft blowing one. I do not, of course, minimize the gravity of those faint blowing murmurs with irregular and uncertain heart's action, which accompany certain cases of myocarditis with but slight endocardial involvement, but I certainly do not prefer a loud rasping murmur to a faint one.

The final course of cardiac cases is always of great moment and interest. The patients in which the disease begins before twelve years have a very important period before them. The physician who ignores the gravity of a cardiac murmur and lightly says that the child will grow out of it, is taking almost a criminal risk. It is quite true that during this period of change some children do materially change for the better. I have during the past year seen two such satisfactory cases —one a boy and one a girl. A murmur had been left, following an acute endocarditis and I had concluded that in neither case was it likely to disappear. Much to my satisfaction, however, the murmurs have disappeared and the hearts give evidence of being in normal condition. The myocardium, it is true, had in both cases not been apparently involved at any time and the compensation had been excellent.

Results like this, however, are not often to be expected and should never be relied upon. The unfortunate opposite in my experience has been more frequently the result. Several children at this trying period have done badly and succumbed even without repetition of the acute endocarditis. On the whole, I should say, when a murmur has been left and the myocardium has been in fairly good condition, children have gone through this critical period without material changes in the cardiac condition. If they go to eighteen or twenty with a reasonably good cardiac muscle, they have a very good chance of going through the next three decades without cardiac symptoms. When the degenerative changes begin to appear, however, in later life, the individuals with impaired heart have not the chance possessed by normal subjects. They more readily succumb to acute disease and to arterial changes, and are not as apt to live to advanced age.

In general terms my experience leads me to agree with Dunn, that the earlier the primary endocarditis occurs the better is the ultimate prognosis, because of the more perfect adaptation of the child to the heart and the heart to the child. They grow up together and are more apt to adapt themselves to each other than they can do in later years.

In the matter of prognosis, I am not willing to agree with the proposition that the number of murmurs is not to be taken into consideration. The patient with a mitral regurgitant murmur alone, other things being equal, has a much better outlook than the one with a double mitral murmur, and vastly better than the one with an aortic murmur. An aortic lesion is a very dangerous thing in a child, but aortic insufficiency is fortunately rare in children. The human pump is like any other pump. A leak in a valve reduces its efficiency. If there is also obstruction to the flow of the fluid, the impairment is increased and two damaged valves are assuredly worse than one. This is a simple mechanical proposition which cannot be effaced by any amount of statistics. Two murmurs are more serious than one and three are more serious than two. There is too much tendency in statistical papers to make out tables based on one element alone. We cannot safely do that in practice. It is true that a heart with three murmurs and a good muscle may be found acting better than a heart with one murmur and a poor muscle. Prognosis based on one element alone will lead to error, and tables based on one element alone will lead equally to error.

The evil effects of anemia upon the child with cardiac disease can scarcely be overstated. It is a contributing factor to weakness and fatty degeneration of the heart muscle. If uncorrected it may be a factor in turning the scale against the child. This is true even of young children, and particularly true in the period after fourteen. The chlorotic type common to girls of that period should be watched for and should be vigorously combated by diet, moderate outdoor exercise, and medication.

The sources of infection of acute rheumatism and the portals of entrance are still somewhat uncertain. The evidence against the tonsils, however, is very strong. The reasons for the removal of infected tonsils, therefore, in a rheumatic child, whether he have cardiac disease or not, are vastly greater than in any other constitutional type. The child with a cardiac lesion who is subject to tonsillitis should certainly have that dangerous portal of infection closed. In my experience, the type of tonsillitis marked by clear, white, pearly spots and a throat comparatively clean, has not been as serious as that type marked by a

grayish exudate upon a soft, spongy-looking tonsil. Two of my patients a few years ago developed endocarditis in the same week with that type of throat. Cultures were taken primarily in the suspicion of diphtheria. No diphtheria bacilli were found, however, but quantities of streptococci. I have come to dread that soft, spongy type of throat with smeary exudate almost as much as an articular rheumatism.

The statement is so frequently made that quinsy is the type of sore throat most closely associated with rheumatism that there must be some truth in it. I have had in my practice many quinsy subjects and have watched them carefully, but can only say that I have been unable to demonstrate any relationship between quinsy and rheumatism, and certainly none between quinsy and heart disease.

The effect of endocarditis on the growth and development of a child has been a question of considerable interest to me. There are certain ones of the more grave cases, particularly those of decided rheumatic tendency with recurring attacks, which do not develop well. It has seemed to me, however, that the constitutional tendency of the child and its rheumatic involvement were often the factors more important than the heart lesions. I have seen child after child with distinctive blowing murmurs develop in a perfectly normal manner. I saw a little girl a few days ago who went through an acute endocarditis accompanying an inflamed ankle at six years, which left a loud, harsh murmur. She has done perfectly well, however, and began to menstruate at twelve years and five months. Now at thirteen years and four months the murmur is but slight; she is developed beyond the average girl of fourteen and is heavier and stronger than her twin brother, who is a perfectly sound boy. When the compensation is good there seems to be no reason why the child should not go on and develop in a normal manner.

The treatment of endocarditis divides itself into two very distinct stages—that of the acute and that of the chronic condition. I have become more and more impressed as the years have passed with the importance of absolute rest during the earlier stages. By rest I mean literally absolute rest in bed, that the heart may have as little labor to perform as possible. We treat a broken bone with absolute quiet and an inflamed eye by removal from the light. We can even put the ulcerated stomach at rest. With the heart, however, the rest must be comparative, but it should be as near perfect rest as we are able to attain. It is not sufficient to keep the child in bed during the febrile stage alone.

Six weeks is the shortest time that any endocardial case can be safely permitted to leave the bed, and it is very rare that it is safe within two months. In fact, I would arbitrarily place the time at two months and beyond that be guided by symptoms. Two symptoms are of especial value in determining this important question of leaving the bed, namely, rapidity and regularity of the heart's action. I do not consent to the child's leaving the bed, whether it be six weeks or six months, until his pulse is well below 100, and is regular. Even then if it becomes irregular or unduly rapid upon moderate exertion I insist upon continuing the quiet. The rapidity on exertion, however, must be looked upon with some judgment. A child who has been several weeks in bed is certain to show rather more rapidity of pulse than one who has been leading an active life, and it is often a question how much importance should be attached to increased frequency of the pulse in the first efforts, if it continue regular. A child should be allowed to sit up in bed and engage in pastimes which will use the arms and involve some physical effort before he is allowed to actually leave the bed.

The management of the acute stage in ordinary cases has been with me a comparatively simple matter. The education of the family has often been more difficult than the treatment of the patient. But family training is necessary. In this particular I have frequently had the experience, especially in the chronic stages, that Dr. Dunn speaks about: the families are prone to go to one extreme or the other. They either ignore your advice and look upon the doctor as an alarmist, or they restrict the child unduly in safe and desirable activity.

After the child is out of bed with a murmur left behind and the chronic stage entered upon, many difficult problems present themselves. The management then often settles itself to one of judgment alone. Text-books give but little help as a rule, and the questions that arise must be decided largely by experience and reason. I am more and more convinced that to maintain the restrictive treatment too rigidly and too long after the preliminary stages is an error. Too much confinement to the house and restriction of muscular exercise may defeat its own object. We are now in a stage where nutrition should be maintained at its highest possible point; where muscular development should be encouraged, and the child prepared to perform its ordinary duties in life. The management of girls is usually easier in this regard than that of boys. Skipping the rope with little girls; dancing, lawn tennis and basket ball with the older ones are the exercises which are most

likely to be productive of harm. With the boys who develop well and grow strong and athletic, the question of athletic games is a very difficult one, and I do not feel that general rules can be given. Here, as in the earlier stages, the rate and frequency of the pulse offers us the best guide, provided the patient is in a reasonably good physical condition. Action of the heart under exercise is the most reliable guide. If it becomes irregular or unduly rapid, there should be no hesitation in forbidding the more strenuous exercises.

A period of daily rest is of great importance to the cardiac child. This can usually be enforced more readily with girls than with boys. Even a half hour of relaxation during the day, whether the patient can sleep or not, is of great value. If that can be secured, we may feel warranted in granting considerably more leeway in the matter of actual exercise.

After the acute stages of endocarditis are passed, we have to deal with a condition rather than a disease. In the management of this condition, arbitrary rules may do harm rather than good. In few other conditions is it more essential that we study the case and treat the patient rather than the disease.

DISCUSSION

DR. CHAPIN: I would like to call attention to one feature of the heart in children, that may have some bearing on this address, on which the pediatrist should have somewhat to say; and that is, the tendency to quick and acute dilatation of the heart of the child under great stress.

Now, young athletes, with or without heart trouble, are allowed to go through tremendous strains as a result of which I believe and know permanent damage is done to the heart. The physician is never consulted; the athletic trainer is the one who is supposed to know all about it; when, as a matter of fact, he is very ignorant concerning these conditions. I had a boy sent to me from one of the leading preparatory schools — a boy of about 12 — who was the leading athlete of the whole school; and after the half-mile race, which he won, he fell down, was apparently unconscious for awhile, was carried into the gymnasium, and the next day I was sent for to find out what was the matter. I found an acute dilatation of the heart that probably will be permanent. The pediatrist should have more to say about these strenuous exercises that the preparatory schools and the colleges encourage.

DR. BOWDITCH: I would like to confirm what Dr. Chapin has said; and, further than that, to connect his remarks still closer to Dr. Hamill's address. I cannot see exactly, under present conditions, how public school children that are allowed to go regularly with damaged hearts are going to have a fair show. There are quite a number of them. It seems to me that closer supervision, closer study of these cases by the school physician or a group of physicians in connection with the teacher would be of great assistance. I should say that such a committee as Dr. Hamill suggests could take up this one piece of work with great advantage.

It struck me it might be interesting if we could see a little bit further into our cases of endocarditis by applying the test that Dr. Richard Smith of Boston has applied in a small number of cases, in seeing how far the family incidence plays a part; how many cases of endocarditis occur in a given family are associated with sore throat and other cognate troubles. Dr. Smith presented quite an interesting short paper before the New England Pediatric Society on the subject, and opened my eyes to the possibility of the family incidence in endocarditis.

DR. TALBOT: Dr. Crandall said that all the children in private practice have better opportunity and treatment than the children of the poor. I agree with that, and am sure that the condition can be remedied. In fact, we have done a great deal toward remedying it in Boston. We have run a heart hospital for three years and have had some excellent results. The children are kept in bed for from six weeks to six months; and I think that from six weeks to two months is too short in the majority of cases. We are now studying in our clinic at the Massachusetts General Hospital what a social worker can do in keeping the children in bed at home; and we hope in two years to be able to report on the efficiency of the two types of work in caring for hospital patients. I am prepared to say now that they are both efficient; but which is the best I am unprepared to say; and we feel that a great many of these children can be cured in the home and that their mothers, no matter how ignorant they are, can give them intelligent care, when they receive individual attention.

DR. GRIFFITH: If I understood Dr. Crandall correctly, it was to the effect that increase in the irregularity of the heart's action on exercise was an indication for continued rest in bed. I think that this is true, but with certain modifications. The *increase* in irregularity is of importance as a guide, but the *presence* of irregularity while the child is still in bed is not always a good reason for its continued confinement there. There are very many conditions, besides and including diseases of the heart, in which there is the existence of persistent irregularity; and a tendency in all young children to a certain degree of this is well known. I have certainly known children to be kept in bed much longer than they ought to be, merely on account of a slight irregularity of the cardiac action. The prognosis of endocarditis in children is subject to much variation, and is influenced by factors depending on the age. It is more favorable in the power of the child's heart to adjust itself to the demands made on it. It is less favorable on account of the greater likelihood of the recurrence of endocarditis at this period of life. We could divide cases of endocarditis in children in three classes. The first, a comparatively small one, of absolute failure of compensation, not much influenced by treatment. A second is a large class, different from adults in that the patients respond well to treatment and in which attainment of compensation usually readily follows. The third is a very discouraging class in which the most apparent symptom is the persistence of anemia in spite of the employment of any measures to combat this. These children are not recognized as subjects of heart disease until a physical examination is made. They show merely a tendency to indolence, a lack of desire to take any exercise, a disposition to engage in quiet plays, and the like. The boys have learned of themselves that they cannot keep up with the active running and exercise of other boys, and quietly adapt themselves to this condition. The anemia which is present is, as I said, one extremely difficult to remove, and from this point of view the prognosis is discouraging.

DR. BLACKADER: The influence of endocarditis on the development of the child is a point which has had much interest for me. When the lesion is severe, development is checked owing to defective nutrition, but if the circulation is not seriously impeded, growth may be more rapid than in the normal child. Dr. Jacobi, many years since, pointed out that growth in childhood was associated with wide arteries, and low blood pressure, and was checked by all conditions which unduly increase blood pressure. In children with serious cardiac involvement, nutrition suffers and growth is hindered; but in children with only slight endocardial trouble in whom low blood pressure persists for many months, and nutrition is not seriously interfered with, growth in height exceeds the normal average. Another point to which I may be permitted to refer is that in cardiac affections in children small doses of potassium iodid, persisted in for a period of some weeks after the acute symptoms have subsided, have frequently appeared to me to have a very definite value in lessening the induration of the affected valve. Again, in children with a moderate mitral lesion, the necessity for prolonged rest is to be emphasized for many months, yet in time a certain amount of freedom in exercise is to be permitted. The persistent checking of a child's impulses to avoid strain on the heart, in my opinion, is distinctly harmful. While the more strenuous games of football and other severe competitive games are to be forbidden, ordinary games are to be permitted without too frequently calling the child's attention to his heart by examination of any kind.

DR. COIT: I was interested in the speaker's index for the length of time these children were to be kept in bed, namely, that when the child's pulse rate was 100 he would allow him to be up. Since little children are more susceptible to the effect of effort on the pulse rate than those in adolescence and adult life, and since many children without the effects of endocarditis have a pulse rate above 100 on slight exertion, it seems to me important that we should know whether this index should be a pulse rate of 100 while still recumbent or a pulse rate of 100 after they are out of bed.

DR. ABT: One or two points suggested by Dr. Crandall's paper. The first point I have in mind is that I think the aortic lesions are not so rare as are commonly supposed, I mean the primary aortic lesion. It seems to me, as I see an increasing number of cases, both in private practice and in the hospital, that the statement that aortic lesions are rare, is rather refuted by my own experience. Whether that is purely a circumstance, or whether it is a general experience, would be of more than general interest.

Another point that occurred to me was in connection with the occasional murmurs that one hears after great physical effort on the part of children. It seems to me that all children, on account of the very elasticity of the heart, and on account of the strenuous exercise that they sometimes undergo, are subject to more or less temporary aortic dilatation; and occasionally one will hear a murmur — a minute cardiac dilatation — for days.

Another point that refers more particularly to the management. What should be done or advised with regard to exercise? It seems to me that all of us are confronted by that difficult problem: the boy (as Dr. Crandall has stated) has been in bed for a long period and is now anxious to resume his play with the other playmates. It seems to me that no absolute rule can be laid down; that it is largely a matter of the examination of the individual child; that if the heart muscle has returned to its normal or near its normal size, in spite of the existence of the murmur, one may permit considerable

exercise. If, on the other hand, there is considerable cardiac dilatation, one would be rather careful about advising the exercise.

DR. TILESTON: I would like to call attention to the frequency with which mitral systolic murmurs during the course of acute articular rheumatism are due to myocarditis, rather than to endocarditis. Kemp (*Quart. Jour. Med.*, April, 1914) in a large series of cases of acute articular rheumatism found that 22 per cent. presented signs of carditis in the acute stage, these signs disappearing during convalescence; and that 20 per cent. of those who at discharge from the hospital showed cardiac signs (usually a mitral systolic murmur) were normal when examined by him two years later. With regard to the frequency of aortic lesions of childhood spoken of by Dr. Abt, I would concur with his opinion that they are rather common than otherwise.

DR. FREEMAN: The value of roentgenography in the control of cases of endocarditis should receive notice. Valuable information as to the condition of the heart is obtainable by this means, while subsequent photographs indicate well the results of treatment, and I think are perhaps the best indications that we have, for in some cases in which the general condition seems to be improving, we find by roentgenography that the condition of the heart is no better. One such case has recently occurred under my observation. The child came to the hospital and was kept in bed for awhile but later was allowed up. She gained in weight and apparently had a better compensation but had a slight elevation of temperature. A second roentgenogram showed considerably more rounding and bulging on both the left and right side of the heart. She was then put to bed and kept absolutely quiet on her back, and in a few days the temperature disappeared entirely and a more substantial improvement took place.

DR. MILLER: I would call attention to cases we all see, of cardiac murmurs in children, the so-called adventitious murmurs, incidental murmurs, that is, which are not due to endocarditis, but which are extremely difficult to differentiate from true endocarditis. These children are of the class of so-called delicate children, and sometimes have the murmur at the base, sometimes at the apex: otherwise the children are not sick. I have never been able to satisfy myself as to the nature of these murmurs. The children have no anemia, and have no symptoms except the fact that they are rather delicate. We see these cases constantly. The child itself, usually, is nervous and the parents are anxious about the murmur; and yet the murmur appears to be of no significance. I think the best treatment of such cases is to permit the children to run around and exercise, entirely regardless of the murmur; after awhile it seems to disappear.

As to the use of opium in acute endocarditis in children, and adults, too: it is a most valuable drug. In some cases, not only in nervous cases, in which there is sleeplessness and general restlessness, but also when there is dilatation and edema, I have seen small doses of morphin or opium not only produce a beneficial effect on the child, but act as an actual diuretic. Ordinary diuretics may have failed; yet small doses of morphin will often produce diuresis, and there will be a speedy return to normal conditions.

DR. EATON: Dr. Crandall touched very lightly on the question of edema. I think that is one of the most important questions for treatment that we have in these cases of endocarditis; and that long administration of iron in this condition is a genuine food and one of our best helps. He intimated that in the usual run of cases we keep the children in bed too long; and I agree with him

there, and too, that each case has to be treated on its own merits; but sometimes I think we forget that many of these cases of heart distress are of such a nature that the heart can be educated to fulfil all the needs of the individual child. Of course, that should be under the supervision of the pediatrist; but, at the same time, we know how much can be done by the proper education of the heart, beginning with a slight amount of exercise and increasing until the child becomes absolutely useful. Of course, it is a question what will happen in their old age; but such hearts, as a general principle, will last as long as they are needed in the ordinary occupations of life. Dr. Bowditch brought up an interesting question as to the family incidence. A maternal grandmother had a lesion for a great many years, a minor regurgitant murmur; her oldest daughter had one; the oldest son of that daughter has had, to my certain knowledge, two attacks of endocarditis by infection through the tonsils. The second daughter in that family — the mother's daughter — had an endocardial murmur, and has it yet. Her oldest child, a daughter, has had two attacks of endocarditis, one of which probably entered through infection through the tonsil. She has had her tonsils removed, as has also the boy of the older daughter. The third daughter has never to my knowledge had any endocarditis, or any endocardial murmur; but her oldest boy has had an attack; and I saw him yesterday: he is having an attack of endocarditis following an infection through the tonsil. It is rather significant, and one of the things of interest if we can trace them. The effect of the predisposition (if one can use that word) in these children from the grandmother's endocardial murmur, one cannot define, but one feels it must have something to do, perhaps, with the susceptibility of those descending from her.

DR. CHURCHILL: Was the blood-pressure taken in these cases and if so, was it of any value in differentiating between the organic and the functional murmur? Did Dr. Crandall notice any difference in the intensity of the murmur when the child was lying down or sitting, or standing? In the last year I happened to run across two or three cases in which there was a definite, quite loud murmur, systolic, found in the child when lying down, but less intense when sitting up, and still less when standing. We can offset the matter of keeping the children too long in bed by exercise in applied gymnastics and so promoting general nutrition.

DR. LAFÉTRA: I think this family tendency to cardiac disease may be explained, perhaps, in Dr. Eaton's cases, by tendency to the infection of the lymphatic tissues, which is often handed down from one generation to the next; and that a good deal of so-called heredity of cardiac disease and rheumatism — rheumatic disease — is probably due to the tendency of the throat, joints and endocardium to become infected. Bad throats are so readily inherited. Dr. Churchill brought up a point with regard to blood pressure, which Dr. Hoobler and I have been investigating; and it does seem that blood-pressure along with the pulse rate can be used as a guide to tell when to allow patients out of bed. I think there is a point that we might take up and investigate in our hospital work, because if, to maintain a satisfactory blood pressure, it is necessary for the heart beat to become more rapid there must be some figure at which, for each given age or weight, we must be cautious about letting the child up. Even when we decide to let the child up at first we may find that the pulse is too rapid, but after a few days of regulated exercise the heart gets its second wind, and then can go on perfectly well.

DR. HAMILL: Did you find wide variations in the blood pressure of the heart cases — wider than in the normal children?

DR. WILCOX: Our experience on Dr. La Fétra's service at Bellevue Hospital in the treatment of the chronic cardiac diseases in children has demonstrated the fact that a great deal of the work done on our wards among this class of patients is productive of only temporary, incomplete and unsatisfactory results. There is something more needed than rest in bed and medical treatment to bring about permanent improvement in these young hearts which are the subject of acute inflammation or have recently recovered from attacks of decompensation. Digitalis, caffein and such cardiac aids accomplish their work in steadying or slowing down the irregular overacting heart. Rest in bed and proper nourishment, even more than drugs, is effective in bringing these patients out of their acute distress, and in restoring the balance of their circulation. When this has been done, however, too often the child finds himself able to support life comfortably only so long as no muscular exertion is demanded of him. It is our common experience that in a short time after discharge, decompensation has recurred and the patients again present themselves for treatment; the rapid, irregular heart is again slowed, errors of digestion are again corrected and once more the patient is discharged barely able to move slowly about and this only for a short time. To provide a means for accomplishing more permanent results in these cases, we two years ago, established at Bridgeport, Conn., what we term the convalescent branch of the Bellevue Children's Service, where we have been sending our cardiacs as soon as they recover from their more acute symptoms, in the hope that the addition of sunshine, fresh air and release from the environment of the hospital ward, added to proper medical supervision and the necessary medication would bring about more permanent results. One thing that these cardiacs all show in common is anemia. This, it seems to me, is a basic condition which, if untreated, prevents proper nourishment to the heart wall and stands in the way of accomplishment of any definite, permanent increase of cardiac efficiency. We may not be able to replace destroyed muscle fibers or to correct lesions of the pericardium or endocardium, but this anemia represents a very important factor in the disease and one which is amenable to treatment. If there is a difference between the results obtained in the treatment of cardiac disease in private and in the hospital, it seems to me that it depends on the difference in environment which is possible to provide for the child in comfortable circumstances as opposed to that available for the hospital cases. Our hope then was that by providing the ideal surroundings furnished in this home we would substitute for the temporary relief heretofore given, in some cases at least, more permanent improvement. The results which we have obtained in these two years have quite borne out our expectation. Twenty-one children suffering from severe cardiac embarrassment were sent to the home and spent from one to three months there. In one instance death occurred during the summer. The other children improved rapidly and maintained their improvement. Throughout the time they were at the home no case of relapse occurred. Although an epidemic of diphtheria in the home involved several of our worse cases, we had no further fatalities.

Through the visiting nurses of the social service department most of the children were kept under observation and returned for inspection after leaving the home. All of these children on leaving the hospital for the country were seriously incapacitated by their disease; all were anemic, or undernourished, some in a severe state of malnutrition; many of them showed the more serious evidence of cardiac failure. It is a strong argument for the value of such outdoor care for these patients that they all walked to the dispensary and in

two instances only, was the cardiac condition such as to warrant the return of the patient to the ward. It seems to me that such measures aimed at the anemia of cardiac disease, are the ones most likely to given permanent results. Of all the secondary symptoms observed in cardiacs, anemia is the common one whose nature makes it most amenable to treatment. Prolonged treatment of young cardiacs in a hospital ward has certainly proved unproductive of permanent results on the condition because of the inevitable anemia which must ensue. It is my belief that, if such outdoor treatment could be provided for these children, a great number of them could be led up to a point in their cure which would make it possible for them to live in comfort and later to carry on some limited but useful occupation.

DR. CRANDALL: It seems to me that this work that Dr. Talbot and Dr. Wilcox have spoken of, is one of the most real charities that one can think of. These children kept in bed too long in a hospital will get worrisome. If they are sent out they go to the tenements and there they have no help and are prone to relapse. When a patient in a cardiac case is doing well let him go home and not call his attention too much to the heart. The pulse rate of about 100 is always significant. If it is a child that you are on good terms with and does not get nervous when you come in you can judge somewhat of his condition, but should attempt to see him asleep.

TREATMENT OF RECURRENT SIBILANT BRONCHITIS

B. K. RACHFORD, M.D.
CINCINNATI

It is my belief that the symptom groups commonly described in medical literature under the titles, migraine, recurrent vomiting, recurrent sibilant bronchitis, recurrent coryza, asthma and urticaria, as they occur in children, are, as a rule, closely related food intoxications which can be successfully treated by very much the same dietetic and medical treatment.

In a paper[1] read before this Society in 1897, I expressed very much the same views that I am reiterating to-day. In that paper under the title "Lithemic Vomiting," I reported cases which are now known as recurrent vomiting, and called attention to the fact that recurrent vomiting attacks may not infrequently, as the child grows older, be transformed into attacks of true migraine. In the seventeen years which have intervened I have become more and more convinced that the clinical statement I then made is true. In that same paper I spoke of the toxic dyspnea which may be present in these cases. I also described in these same cases acute gastro-intestinal attacks associated with diarrhea, which recur without apparent cause at more or less regular intervals, and hazarded the belief that the diarrhea in these cases was eliminative and therefore a curative symptom. At the same time I also expressed the belief that urticaria might be an expression of these same auto-intoxications.

In 1899 Dr. Frederick A. Packard, under the title "Urticaria of Mucous Membranes," presented to this Society the then existing clinical evidence of the mutual relations of asthma and urticaria.

Dr. Charles Gilmore Kerley has for a number of years been especially interested in the close etiological relationship which exists between the above named symptom groups and has insisted that the treatment of all of these disorders should be along similar lines.

It is not my purpose at the present time to express an opinion as to the character or the method of action of the various toxins which

1. Rachford, B. K.: Trans. Am. Ped. Soc., 1897.

may bring about the above named symptom-groups. The special objects of this paper are:

First. To call attention to the clinical fact that recurrent rhinitis, and recurrent sibilant bronchitis, which are sometimes associated with rather severe asthmatic attacks, and almost always with a severe paroxysmal cough, are very commonly caused by some kind of food intoxication.

Second. To direct attention to the fact that recurrent attacks of rhinitis and whistling bronchitis, which is one of the most common symptom-groups the physician is called on to treat, may be, in most instances, almost or quite relieved by dietetic treatment.

Third. To insist that this symptom-group occurs not infrequently in children who have a predisposition to migraine, recurrent vomiting, and urticaria. In the case histories given below this relationship is shown.

The successful treatment of these various syndromes is very similar and varies but slightly from that which I recommended in 1895[2] and 1903.[3]

TREATMENT OF RECURRENT RHINITIS, RECURRENT SIBILANT BRONCHITIS AND ASTHMA IN THE CHILD

Dietetic Treatment of Attack.—During the attack sweets, butter-fats, eggs, and raw fruits are especially to be avoided. Strawberries, rhubarb, tomatoes, salads, shell-fish, tea, coffee, pastry, gravies, cream, cod-liver oil and alcohol are excluded from the diet. If it be necessary to sweeten the cereals and cooked fruits which the child eats, saccharin is to be used for this purpose instead of sugar, and while the patient is not to have butter, skimmed milk may be allowed. Eggs in every form, even in cooked foods, are to be carefully eliminated from the diet, and among the raw fruits, oranges are to be especially avoided.

The following foods may be allowed: beef, mutton, fowl and fish in moderation, cereals, bread, all vegetables not above proscribed, cooked fruits, skimmed milk and thick soups.

Interval Dietetic Treatment.—About two months after the patient has recovered from the attack the above diet may be carefully modified

2. Rachford, B. K.: Med. News, Sept. 7, 1895.
3. Treatment of Migraine, Trans. Assn. of Am. Physicians, 1903.

by adding one egg a day to the diet, then perhaps two weeks later, milk containing 4 per cent. fat may be substituted for skimmed milk. Some few weeks later all raw fruits, oranges excepted, may be added to the diet. Some weeks later small quantities of sugar may be used for sweetening cereals and stewed fruits. After five or six months the child may return to his original diet, with the exception that he must eat sparingly of sweets and must eat nothing between meals. If the symptom-group returns on the addition of any article of diet, that particular food is to be permanently excluded from the diet.

Hygienic Treatment.—During the attack the patient is to have all the outdoor air he can get, with as little exercise as possible; this can be obtained on sleeping porches and with wide-open windows. As he recovers from his attack, exercise in the open air is advisable. It is especially important that schoolchildren who must necessarily spend a portion of their day in the close atmosphere of the schoolroom should have all of the air possible at night.

Medical Treatment of the Attack.—During the attack, constipation should be relieved by some of the magnesium salts.[4] The milk of magnesia or calcined magnesia are especially suitable for this purpose. Sulphate of magnesia may be used in small doses in older children.

Alkalies in the form of bicarbonate of soda, bicarbonate of potash or citrate of potash should be given in fair sized doses three or four times a day. Bicarbonate of soda may be given, for example, in 5-grain capsules three or four times a day, or the same size dose of citrate of potash may be given in solution. In younger children who object to the taste of bicarbonate of soda I commonly direct the mother to distribute through the child's food a teaspoonful of this salt each day.

Tincture of belladonna should be given for a short time in 2 to 4 minims doses three times a day.

Interval Medical Treatment.—If constipation cannot be controlled by diet, some form of cascara or other palatable laxative should be used as indicated. The belladonna is discontinued, but an alkali in some form should be given for six or eight weeks. Bicarbonate of soda is the best alkali for this purpose and may be given in moderate sized doses once or twice a day.

4. Martin Fischer has taught us that the magnesium salts when given with such alkalies as soda and potash help to fix these alkalies in the tissues.

The above treatment if faithfully carried out will in almost every instance control the syndrome above described. This treatment is almost or quite as effective in the treatment of migraine and recurrent vomiting in children. In the adult and older child I continue to use in the treatment of migraine the formula[5] which I originated twenty years ago, and which has been published many times. It is as follows:

Sodii sulphatis (dry)	30 grains
Sodii salicylatis (from wintergreen)	10 grains
Magnesii sulphatis	50 grains
Lithii benzoatis	5 grains
Tincturae nucis vomicae	3 drops
Aquae destil. to make	4 ounces

This prescription is put up in siphons and charged with carbonic acid, and the patient is directed to take, half an hour before breakfast, a sufficient quantity to produce at least one bowel movement during the morning. This prescription is a remedy of great value in the preventive treatment of migraine; it may in fact replace all other medication.

CASE REPORTS

CASE 1.—Male, aged 10. Family history good, except mother has migraine. Two other children are well. Patient began to have attacks of recurrent vomiting when two years of age. For a few years these attacks were frequent and severe and were accompanied by a severe acidosis. Acetone and diacetic acid were found in the urine on first day of attack and continued until after the vomiting ceased, somewhere between the fifth and ninth day of the attack. Up to the time he was 5 years of age these attacks grew more frequent and more severe and his general physical condition was far from satisfactory. He was thin, undersized and lacked strength. He suffered frequently from rhinitis and bronchitis. After 5 years of age his attacks of recurrent vomiting were less frequent and less severe. Between 7 and 10 years of age he did not suffer from recurrent vomiting but during this time he had frequent attacks of bronchitis characterized by severe coughing and by whistling râles.

Sept. 15, 1913, he became ill with severe coryza, mild pharyngitis and whistling bronchitis. He had fever and was put to bed. He was given 5 grains of potassium citrate and 2 minims of tincture of belladonna. For six weeks he continued to manifest the symptoms incident to coryza, pharyngitis and bronchitis. At times he had no fever, little cough and seemed on the point of convalescence, when suddenly without apparent cause there would be an acute exacerbation of all his symptoms and he would go to bed again as ill as before. During this time he had careful local treatment of the nose and throat and such internal treatment as his general symptoms demanded. The internal treatment consisted largely of tincture of belladonna and some alkali. November 1 he suddenly developed a violent urticaria. His body was covered with large

5. Diseases of Children, Rachford. D. Appleton & Co., 1912.

red urticarial wheals. He suffered intensely from itching, he was sick at his stomach, his breath had an acetone odor and his urine contained acetone and diacetic acid. Coincident with the development of the urticaria, his coryza, pharyngitis and bronchitis disappeared.

As I studied the various symptom groups which this patient presented during his illness I became convinced that he was suffering from some kind of food-poisoning and that these poisons, whatever might be their nature, by their action on different parts of the vasomotor nervous system produced from time to time the symptoms of recurrent vomiting, recurrent coryza, recurrent bronchitis and urticaria.

Acting on this hypothesis I placed the patient on the following treatment: I excluded from his diet all sweets, including sugar, all butter fats (including milk), eggs in every form and raw fruit juices, especially oranges. He was allowed soups, cereals, meat, bread and vegetables. The medication consisted of a teaspoonful of bicarbonate of soda every four hours and a sufficient quantity of milk of magnesia to keep his bowels open. Under this treatment he commenced to improve at once and was well within the week. From that time until the present writing, a period of four months, he has been kept practically on the same diet and has remained perfectly well.

CASE 2.—Male, aged 8; family history, paternal grandmother died of tuberculosis, father has hay-fever, mother suffers from migraine. Two other children in the family are well and show no tendency to hay-fever, asthma or autotoxic attacks.

Patient is a boy of fair physical development. He enjoyed good health until eighteen months ago, when he developed a whistling asthmatic bronchitis from which he suffered more or less for nine out of the subsequent twelve months. During this time he had more or less severe attacks of asthmatic bronchitis characterized by great difficulty in breathing and incessant coughing; these symptoms were worse at night. From these severe attacks he would, after a short time, sufficiently recover to return to school. But during all of this time even when he was well enough to go to school, he continued to have whistling râles and a more or less irritating cough, with shortness of breath on exercise. He lived in the open air and slept on a sleeping-porch during all of this time. During the summer of 1913 he had an attack of what his parents believed to be hay-fever. During this time, the last three weeks of August, he suffered severely from asthma. Following this attack in August he remained in comparatively good health until the middle of October, when again he suffered severely from a whistling bronchitis associated with an irritable and paroxysmal cough and both night and day his breathing was labored and his chest was filled with whistling râles.

When I saw him for the first time, Oct. 29, 1913, he was suffering as above desribed. With his asthmatic symptoms, however, he had no fever and so far as I could learn his attacks had always been afebrile, except when complicated by influenza during a household epidemic of this disease.

Treatment.—The treatment was the same as in the preceding case—sweets, butter-fats, eggs, oranges and all raw fruits were eliminated from his diet. He was given bicarbonate of soda three times a day and in addition 5 grains of potassium citrate and 2 minims of tincture of belladonna. This boy was allowed skimmed milk, bread without butter, cereals without sugar or cream, all meats and vegetables and certain cooked fruits. His cereals and cooked fruits were sweetened with saccharin.

Six days later this boy was absolutely well and his general appearance was much improved. The diet and bicarbonate of soda was continued and the boy returned to school and continued to sleep as he always had done in the open air at night. He remained perfectly well until Christmas week, a period of about two months, when he went off of his diet, eating sweets, eggs, butter-fats and raw fruit. Within four or five days all of his asthmatic symptoms returned. He was again placed on the same treatment as before and he as quickly recovered, returning to school perfectly well after the holiday vacation. He remained perfectly well again for two months and then again went off his diet with the result that the asthmatic symptoms again returned. These asthmatic symptoms lasted only a few days after he was again placed on his diet. From that day to the present he has continued his diet and remained quite free from asthmatic symptoms.

CASE 3.—Female, aged 4 years. When 5 months old the child had an attack of whistling bronchitis associated with a severe paroxysmal cough and during the attack vomited a number of times. This attack lasted about three days. She had similar attacks at the ages of 13 months, 14 months, 17 months and 2 years. From that time on during the past two years her attacks have been more severe and more frequent. During the winter of 1912-13 she had nine quite severe attacks within seven months and during the summer of 1913 these attacks occurred about a month apart. Last August she had a very severe recurrent vomiting attack associated with a wheezy bronchitis and a severe paroxysmal cough. This attack lasted six days and during this time almost nothing was retained on the stomach. She had similar attacks Sept 20 and Oct. 20, 1913.

Nov. 16, 1913, she began to cough and quickly developed a severe whistling bronchitis with such alarming symptoms that her parents thought she would die from suffocation during the night. On November 17 the cough was somewhat less paroxysmal and the asthmatic symptoms less severe, but at this time she became sick at her stomach and vomited frequently during the afternoon and night of November 17; in fact, nothing stayed on her stomach for twenty-four hours.

November 18 I saw her for the first time and obtained the above history, and I also learned that in the intervals between the attacks above described she was at no time entirely free from cough and had most of the time whistling sounds in her chest.

When I saw her she was suffering from a whistling bronchitis with marked difficulty in breathing, but the persistent vomiting had apparently subsided. The mother made the observation that during her attacks she suffered either from severe vomiting or from great difficulty in breathing. When the vomiting was severe the asthma would be relieved and vice versa. The urine of this patient contained acetone and diacetic acid.

Treatment.—A diet similar to that outlined in the previous cases was prescribed. She was also given milk of magnesia, bicarbonate of soda, citrate of potash and tincture of belladonna. The patient entirely recovered within a few days and from that day the diet has been continued and small doses of bicarbonate of soda given every day. To the present (a period of over six months) the patient has remained perfectly well.

CASE 4.—Male, aged 8 years. This boy has for two years suffered from typical attacks of true migraine. He has recurrent, self-limited attacks of severe paroxysmal headaches, usually unilateral, always accompanied by nausea, vomit-

ing and vertigo. He goes to bed with these attacks and remains there one or two days. These symptoms are followed by a long and profound sleep from which the patient awakes free from pain. The urine during these attacks commonly contains acetone.

During the past few months he has had these attacks almost every week and during the past week he has had three severe headaches. He is slightly constipated, occasionally demanding a laxative. The eyes, nasopharynx and genital organs have been carefully searched and no reflex factors found. On inquiry I learned that this boy eats from two to four eggs a day, drinks three glasses of rich milk, and eats largely of sweets including large quantities of sugar, and has oranges or grape-fruit for breakfast. He eats almost no vegetables, stewed fruit, very little meat except bacon for breakfast, and cannot be induced to take bread without large quantities of butter.

Treatment.—For this patient I prescribed the same diet outlined above. He was to have absolutely no raw fruit, no eggs, no butter and as little sweets as possible. I also prescribed one-half teaspoonful of bicarbonate of soda three times a day and a teaspoonful of milk of magnesia night and morning. His mother thought it would be absolutely impossible for him to get on with the above diet. I, however, insisted and told her if he could not eat the prescribed foods to let him go without eating. Six weeks later this boy, who had not had a moment of illness during that time, was greatly improved in his general appearance. He was eating meat, vegetables, cereals, skimmed milk, bread and cooked fruits. I then allowed him one egg a day and three glasses of certified milk, which contained 4 per cent. fat. Two months later this boy is perfectly well, is satisfied with his diet, has had no headaches and has greatly improved in his general health. He has gone to school all the time. He still continues to take 5 grains of bicarbonate of soda three times a day and an occasional dose of phosphate of soda or milk of magnesia to move his bowels.

DISCUSSION

DR. EATON: I take it, from Dr. Rachford's paper, that his definition, particularly of this sibilant bronchitis, is a bronchitis in which there are many sibilant râles, mixed in with the râles which we are apt to term asthmatic râles.

I think that in many of these cases (our local cases, I mean) we might view these symptoms in the light of the environment of the child, that is, the part of the country in which it lives. Up to last year — a year ago this past winter — Dr. Wood, my associate, and myself saw occasionally a case of asthmatic bronchitis, sometimes with recurrent symptoms.

A year ago last winter I saw ten cases of acute bronchitis in children, of the asthmatic type. This is what Dr. Rachford, I think, would call sibilant bronchitis. We didn't have any suspicion that it was dietetic in origin: it seemed to be an infection of some sort. This past winter of 1914 we have seen somewhere between ninety and one hundred cases in both hospital and private practice; and in three or four cases I recall it was recurrent in its manifestation. The hospital cases we have had examined as fully as possible. We have had blood cultures made; we have had the sputum examined bacteriologically, thinking, because these cases were so absolutely typical, that we had an infection with which we were not familiar, and which was possibly some new infection; and we have discovered no typical organism. The bacteriologic experiments in the hospital have scarcely paid; we have discovered nothing new;

and yet we feel that there is an element of infection which has come to our part of the country which we have not seen before. While in those cases we are always very careful with the diet, and while I think we can say we have always used the magnesium salt as the first eliminant in those cases, we have found out in getting results quickly the best drug is hexamethylenamin, which is used in comparatively large doses, even in the little children. We have had these cases of asthmatic bronchitis or sibilant bronchitis in nurslings, as well as in the artificially fed children, of ages from a few months up to 7 or 8 years, but I think the majority of the cases are among children from 6 months to 2 years of age; and, as I say, the increase in this particular type of bronchitis in our part of the country has been marked — ten cases in the winter of 1912-13; and I am not prepared to say exactly, but somewhere between ninety and one hundred in the winter of 1913-14; I speak of it at length, because it has some characteristics of bronchial difficulty new to us in western Pennsylvania and eastern Ohio; and whether it be due to any specific infection, or whether due to dietetic errors I am absolutely unable to say. The diet question is a new one to me in regard to those cases.

DR. TALBOT: Two and a half years ago I started to immunize a case with egg anaphylaxis, according to Dr. Schloss (*Am. Jour. Dis. Child.*, 1912, iii, 341), when he became cured and could eat eggs, attacks of asthma which he had been having frequently, ceased for good. Cases studied subsequently showed that eggs were a direct causative factor in the production of asthma.

Some of them cannot take small enough doses of egg so that it is safe to immunize them. Some cases of asthma are not due to eggs.

DR. PISEK: I want to emphasize what Dr. Talbot has just said. It seems to me that Dr. Rachford has given us what we may term the palliative treatment, not the cure. The cure lies in finding, in the particular individual, the positive factor (which is in all probability anaphylactic in character), in eliminating that, and setting up an immunization. There is no question but that very often egg is present in the prepared food even tho we have prohibited eggs in the diet. Even such small quantities of egg must often be eliminated. Therefore we should not lose sight of the dietetic treatment to really bring about the cure.

DR. COIT: Since it is necessary sometimes to eliminate egg from such foods as desserts, I would mention the anaphylactic effect of macaroni, which contains egg, in children who have an idiosyncrasy for eggs.

I had an interesting case illustrating the importance of diet in its relation to food poisoning. It was necessary to transfer a baby, which had been fed exclusively on milk, to a second year diet, commencing with cereals. This baby immediately developed marked depression and temperature on the introduction into its bottle of a teaspoonful of wheat jelly. Besides the temperature and prostration, there was considerable vomiting. I did not believe the teaspoonful of cereal caused this sickness, and urged the mother to persist. She did so, with disastrous results. We eliminated the cereal and after two weeks a teaspoonful of wheat jelly was again introduced into the bottle twice in twenty-four hours, with immediate recurrence of depression and fever. I concluded that the mother was right and that the cereal caused the illness. I made several attempts to advance, using a sort of vaccination method of introducing the cereal in the minutest quantities with the bottle. It was six weeks before the baby tolerated the cereal without symptoms.

I asked Dr. William H. Park if he thought I had caused an anaphylaxis in that baby. He said: "I have no doubt of it." This was an anaphylaxis due to a foreign protein introduced into the bottle. The baby is now taking cereal jelly, and vegetables of all sorts are well borne.

DR. CARR: I wish to endorse Dr. Rachford's paper. There is one factor Dr. Eaton brought out, namely, that many of these children are more susceptible to infection. Allowing for the lithemic factor we must not overlook enlarged tonsils, a deviated septum or hypertrophied turbinates. There are also idiosyncrasies in these patients. A child under my care could ride a pony in the park without bringing on an attack of asthmatic bronchitis but if she went into the stable she invariably had an attack. The children who have fever when they develop an acute attack, do very much better if they are put on a starvation diet, just as in the treatment of catarrhal enteritis. Keep the children for a time without milk and when it is resumed begin with either skimmed or a lactic acid skimmed milk.

DR. ABT: I was reminded by Dr. Rachford's paper of the continental description under the caption of "exudative diathesis." This exudative diathesis presupposes a constitutional state, or some deviation from the normal constitution, and is associated with certain definite symptoms; and it seems to me that if we accept this nomenclature or terminology, we can suppress a great many of the difficulties described by Dr. Rachford under this constitutional state: for example Czerny and Keller described a variety of constant symptoms of eczema, and various other eruptions, as scrofulosis infantum and asthma, similar to those Dr. Rachford has described.

They point out, too, that the symptoms are most pronounced after drinking milk, or eating eggs; and that they occur sometimes after mild infection. It seems to me one must not necessarily consider or presuppose a simple anaphylactic process. We know that many children tolerate eggs and milk in almost excessive quantities; whereas, other children of a peculiar type, with a combination of symptoms, will react badly.

DR. KERLEY: I read before the New York State Society three or four weeks ago a paper which was so similar to that of Dr. Rachford, that those who have heard it here must recognize the similarity. This paper will be published later in the *Archives of Pediatrics* and represents my observations on recurrent bronchitis in children.

DR. ADAMS: When Dr. Jacobi and I were boys some years ago and we ate green apples, you know what we had. These cases are very much on the same order. We heard of internal hives. Are these cases internal hives? These are simply idiosyncrasies to quinin, other drugs and foods which the individual finds out and by eliminating these we can, in almost all instances, cure the attacks.

About five or six years ago I developed what the specialists called "vasomotor catarrh." I don't know whether that is right or not; but I know if I take anything with a flavor of pineapple in it that I will have the most infernal catarrh or asthma, which keeps me awake; I look out for and avoid the flavor of pineapple. I have seen these cases of egg anaphylaxis, if you want to call it such; the patients cannot take eggs in any form; and they have the internal or external hives. We must forbid eggs and be careful afterwards to keep that article out of the diet, just as we would keep the person who cannot take quinin from its use.

Last summer a child who could not take egg in any form was treated by my assistant. The mother told the physician that the child could not take egg. He did not heed the experience of the mother. He was fired; and I was also. We are getting back to first principles. We are not groping around in the mysterious, scientific way, but are content with the simple fact that some persons have these kinks, physical or mental, and all we want to do is to take out that particular kink or the article which produces the kink, and we will all be happy.

DR. MILLER: I would like to call attention to one condition which Dr. Rachford refers to rather indefinitely, namely, urticaria or hives. Other than digestive or metabolic processes may be active. There are certain children who cannot live at the seashore because they get hives. The attacks recur so long as they remain at the shore; but as soon as they get away from the shore the hives cease. I have had several such patients. Something in the air appears to irritate the skin, for just as soon as they get inland, particularly if they go to a mountainous district, they are rid of their complaint.

DR. GRIFFITH: If these cases are instances of anaphylaxis to egg albumin, as suggested by Dr. Talbot, and as seemed in some cases very likely, then it was illogical to prescribe a diet of skimmed milk. Anaphylaxis, it is true, might be dependent on the proteids of milk as well as that of eggs; but benefit coming from the skimming of the milk would be, of course, through the removal of the fat; and the benefit from absence of eggs in these conditions might be dependent on the fact that the fat of the egg was removed. Clearly there are two different factors to be considered in this connection.

DR. RUHRÄH: It occurs to me that we are confusing two things: one, the real anaphylaxis, or food idiosyncrasy; and the other, acid intoxications.

DR. EATON: I have now under observation a child of 15 months, who cannot take cow's milk or eggs in any form. A teaspoonful of baked egg custard will cause the child to spit the minute it touches its mouth; and where the custard spray strikes the hand or any part of the face a red spot develops almost instantly. These cases I have described, however, may not be of the same class as those to which Dr. Rachford has referred. The infection in some of the cases which I examined — cases in which a microorganism has been isolated — was the streptococcus, or mixtures of bacteria; but nothing which could be fairly called pathognomonic.

DR. RACHFORD: Dr. Eaton's inquiry brings up in these cases the question of alkalinity and of the use of alkalies in their treatment. In my paper I have called attention to the fact that Dr. Martin Fischer has found that the magnesium salts, when given with such alkalies as sodium and potassium, help to fix these alkalies in the tissues; in other words the alkalinity of the blood is better kept up when soda or potassium is given with one of the magnesium salts than when either soda or potassium are given alone or with some cathartic other than magnesium. Whatever may be the explanation of the physiologic action of these medicines I am convinced that the combination of alkalies here mentioned is of value in the treatment of recurrent vomiting and recurrent sibilant bronchitis.

In my paper I took special pains to avoid the use of the word anaphylaxis as I wished to discuss this group of cases from a purely clinical point of view — stating clinical facts and avoiding theoretical discussions. In my text-book (1912) I used the term anaphylaxis in explanation of the etiology of these cases, but in this paper I did not care to have clinical facts obscured by theoretical discussions.

A CLINICAL STUDY OF TYPHOID FEVER IN CHILDREN

HENRY DWIGHT CHAPIN, M.D.
NEW YORK

The fact that children have not been supposed to be very susceptible to typhoid fever, and, when affected, are apt to exhibit an irregular type of the disease, would seem to render a study of cases of value for future comparison and reference. It is during an epidemic that they are most liable to be attacked, so that such an occasion affords the most favorable opportunity for a study of any peculiarities the disease may show in early life. Since the employment of Widal's test, the field has widened by proving that typhoid fever in certain mild types may be overlooked in children or mistaken for some other affection.

During the fall of 1913, an epidemic of typhoid fever occurred in the lower East side of New York, including 521 reported cases, with a mortality of 11.7 per cent. Many children were attacked as shown by the following reported number of cases: Under five, 36; five to nine, 98; ten to fourteen, 87. These cases, I think, show a much larger proportion of attacks among children than have usually been reported. A little over two-fifths of all the cases in this epidemic occurred in children under 14 years of age. Dr. Osler in reporting on 1,500 cases of typhoid fever in Johns Hopkins Hospital states that 231 were under 15 years. Dr. Ogan of the New York Health Department, in a study of 1,500 cases in 1912, to compare with Osler's series, found 437 under 15 years. In a personal communication to the writer, Dr. Ogan states that children under 12 constitute one-quarter of the population of New York City and that approximately one-quarter of all cases of typhoid fever occur under 12 years, so that children contribute their fair ratio to this disease. It is evident that we must revise our ideas as to the frequency with which typhoid fever occurs in children.

The cases occurred during August, September, October and November and the Board of Health succeeded in tracing the outbreak to infected milk. It was found that 10 per cent. of the people living in the district were supplied by one milk company and that 70 per

cent. of the cases reported were getting milk from one dairy. A case of typhoid fever was found in the vicinity of this dairy, although not directly connected with it, and it is highly probable that the infection was carried by flies. When the milk from this source of supply was cut off by the Health Department, the epidemic ceased. The fact of it being a milk outbreak probably accounts for the large number of children attacked.

The eleven cases here reported were treated in the children's wards of the New York Postgraduate Hospital. The ages were as follows: 2 years, 1 case; 3 years, 2 cases; 4 years, 2 cases; 5 years, 2 cases; 6 years, 3 cases; 8 years, 1 case.

The duration of the fever, including the period before admission, as given in the histories, was varied and ran, from the shortest to the longest period, as follows: 7 days; 11 days; 16 days; 18 days; 19 days (two cases); 20 days; 21 days; 24 days; 33 days; 42 days. In only five cases did the temperature run very high during the active stage, two reaching 106 F., one 105.2 F. and two ranging from 104 F. to 105 F. The temperatures were not high in the other cases and were distinctly remittent toward the close of the disease. The diagnosis was confirmed in every instance by a positive Widal and no case showed any rose spots.

A study of the blood on admission is shown in the following table:

TABLE 1.—RESULTS OF BLOOD EXAMINATION ON ADMISSION

Age Years	Reds	Whites	Polymorphonuclears Per Cent.	Lymphocytes Per Cent.
8	5,200,000	7,200	58	42
5	4,028,000	5,700	65	35
3½	4,352,000	10,000	81	18
4	4,088,000	4,200	52	48
2	4,130,000	9,100	68	32
3	4,270,000	13,700	68	32
6	4,848,000	9,200	82	16
6	4,600,000	7,400	64	36
4	4,800,000	10,100	74	26
6	4,264,000	8,700	79	21
5	4,600,000	6,000	64	35

An inspection of this table shows the red corpuscles to be about normal in numbers. There is no appreciable diminution of the leukocytes except in one case, a child of 4 years, in whom they numbered

only 4,200. These cases thus failed to show the leukopenia that is supposed to accompany typhoid fever. In the differential count, a study of the polynuclears shows what may be considered a polynucleosis when we have in mind the age of the patients. In every instance the percentage is appreciably higher than the average for the age. The two most marked were in a 3-year-old child showing 81 per cent. against a normal of 42 per cent., and a child of 6 years with 82 per cent. against a normal of 52 per cent.

The lymphocyte count was more irregular, being normal in only one case, a child of 6 years, with 36 per cent. In two cases, the count was above the normal for the age, one child of 8 years giving 42 per cent. against 33 per cent. average, and another of 4 years showing 48 per cent. against 41 per cent. average. The remaining eight were below the normal for the age. In two of the cases the relative diminution was very marked, one child of 3 years having only 18 per cent. and another of 6 years 16 per cent. against 47 per cent. and 37 per cent. averages for these ages.

A study of the urine showed that the kidneys were not much affected in this series; six cases were entirely negative, three gave traces of albumen, with occasional hyaline and granular casts, and a small number of pus cells were also reported in four of the cases.

The gastro-intestinal symptoms were neither marked nor severe. Vomiting was noted at the onset in four cases and during the fever in one case. Five of the patients had diarrhea, which was more occasional than constant; five were constipated most of the time, and one showed no abnormality. They seemed both to enjoy and digest the generous dietary that was given. For a number of years the opinion has been gaining ground that typhoid fever patients were being fed on a too exclusively weak and liquid diet. While the latter was based on the idea that a bland, fluid diet was safest for an inflamed, irritated bowel, the loss of weight and extreme inanition that sometimes followed this plan had unfortunate results. Some patients cannot take sufficient milk or other fluid nourishment to repair the waste caused by the fever and keep up the general vitality. It must be remembered that all digestible food is in liquid form when it passes from the duodenum into the ileum, and that feces, consisting largely of dead bacteria, epithelial cells and the secretions of the bowel, usually become solid only in the lower part of the colon. It is difficult to see how such material can have any appreciable effect on ulcers usually located

in the ileocecal region. A more generous and varied diet not only stimulates the jaded appetite and digestion, thus making the patient able and willing to take more nourishment, but the resisting powers are thereby increased and the dangers of relapses thus lessened.

Acting on this idea, the children were allowed the general ward diet with the exception of meat. This included milk, cocoa, eggs, bread, toast, crackers, cereals, jelly, potatoes, gravy, broths, custard, junket, apple sauce, orange juice, ice-cream and lady fingers. They received nourishment once every three hours throughout the day.

An effort was made to give them forty calories per kilo body-weight per day, but in not a single instance was it possible to make them take the full required number of calories through the febrile stage. In some instances, sugar of milk or malt-sugar was added to the liquids in order to increase the total caloric value of the food but this also failed, since the children refused to take it. Dr. Dennett, who had this feature of the work in charge, concluded that while from a theoretical standpoint it would be beneficial to have them take the required number of calories, it was not practicable to carry out the caloric feeding in these cases, since children, unlike adults, cannot be forced to take food.

The success of this varied diet is shown in Table 2.

TABLE 2.—DATA CONCERNING TYPHOID PATIENTS ON LIBERAL DIET

Age Years	Weight			
	On Admission Pounds	On Discharge Pounds	Gain Pounds	Loss Pounds
8	43¼	41⅛	....	2⅛
5	30⅝	32¾	2⅛	
3½	31⅛	33⅞	2¾	
4	29	32⅝	3⅝	
2	24	23¾	....	¼
3	22⅜	30⅜	8	
6	40⁹⁄₁₆	38⅜	....	2³⁄₁₆
6	33	34¼	1¼	
4	22⅝	30¼	7⅝	
6	43⁵⁄₁₆	45¼	1¹⁵⁄₁₆	
5	37⅝	37¼	....	⅜

It is seen that seven cases gained in weight during the fever, two showed a very slight loss and two an appreciable loss. The most

remarkable cases occurred in a child of 3 years who showed a gain of 8 pounds after a fever lasting nineteen days, and another of 4 years gaining over 7 pounds after a fever of twenty days.

There were no very severe complications and no relapses. Under the former may be noted two cases of tonsillitis, two of otitis media, one hypostatic pneumonia, one pyelitis and one severe bronchitis.

Without a knowledge of the epidemic and the use of the Widal test, it is doubtful if most of these cases would have been properly diagnosed. In no case was the spleen palpable. They came in with the following diagnoses: Probable typhoid fever, 5 cases; Brill's disease, 1 case; meningitis, 1 case; miliary tuberculosis, 1 case; bronchitis, 1 case; no diagnosis, 2 cases.

I am indebted to Drs. Dennett and Steinmetz for aid in watching and collating the cases.

DISCUSSION

DR. HOLT: How long a period does Dr. Chapin refer to during which the patient gained eight pounds in weight?

DR. CHAPIN: It meant on an average from three to four weeks.

DR. HOLT: Those that gained most?

DR. CHAPIN: They were discharged from the hospital a few days after the fever left them.

DR. ADAMS: Some few years ago I analyzed 550 cases of typhoid fever that had occurred in the Children's Hospital under the observation of Dr. Acker and myself, in which I took up all — and more — of the points mentioned by Dr. Chapin. The records of this society will show that some twenty years ago a prominent member stated that in a number of thousand necropsies performed at a New York institution not one case of typhoid fever in infants had ever been found. Shortly after that I presented a specimen from a child in the Foundling Hospital, in Washington which had died of intestinal hemorrhage, in which the evidences of typhoid fever were undoubted.

A few years ago, at the invitation of Dr. Rotch, I gave a talk to his class at the Harvard Medical School on "Typhoid Fever in Infants Under Two Years of Age." At that time there was still some doubt as to the occurrence of typhoid fever in children under 2 years of age. Shortly after that, my friend Dr. Morse wrote to me that he had had, I think, two cases within a very short time, in which the diagnosis had been made in infants under 2 years of age. I have seen within the past two years two nursing infants each of which had contracted typhoid fever from its mother.

One thing which has impressed me of late in the treatment of typhoid fever is the absence of the wailings and the pitiful sights that we did see in the children's hospitals and in private practice of children begging for food. Go to my wards at the present time and ask the children with typhoid fever if they are hungry. They are never hungry. In private practice the same thing is true. I quite remember in my own family my child begging for food, crying for food, when fed on the slop that was given in those days. The child, when

she hears of patients with typhoid fever to whom I have given eggs, mushes, et cetera, says: "Father, why didn't you give me those things when I had typhoid fever?" We knew that the child had to be supplied with a certain number of caloric units in twenty-four hours and that if we didn't supply artificially those units the child must take it from his own person: in other words, he must eat himself up; and yet we went along year after year starving these patients and increasing, instead of decreasing the mortality, by starvation; and it was not until Shattuck and men of his stamp came forward and advised the increase of diet that men (and even I remained skeptical for some time) took up the method he advised. Every one who has practiced the modern method of cutting out the slops and drinks supposed to be nutritious and feeding the children on an enriched food in which they get at least a sufficient number of caloric units in the twenty-four hours to support them is convinced of its value. Typhoid fever has never been, among children, a rare disease in the District of Columbia. Busby, long before I could enunciate an opinion, recognized the disease in young children; W. W. Johnson, in Washington, recognized it in the '70's; and yet, the profession at large were loath to accept the fact that a child could have typhoid fever. The youngest baby that I have ever seen with a typical attack of typhoid fever was between 5 and 6 months old, the disease running along for four or five weeks and conforming to the severe type.

DR. LAFÉTRA: My paper, of course, bears very much on this topic; so that naturally I cannot now allude to a good many of the things I will say in it; but just one word with regard to the number of calories that can be taken. I was surprised that Dr. Chapin had difficulty in giving his patients as many as 40 calories per pound. At Bellevue Hospital we found that when the children first entered the wards they would take very little food; but just as soon as they began to eat at all, they would take the ordinary 40 or 50 calories very readily, and very soon were taking two or three times that amount and with great benefit.

DR. JACOBI: There are cases on record of typhoid fever in newly born and in very young babies. One of them has undoubtedly been forgotten; because it occurred about thirty-five years ago. That was in my own practice. The baby died of typhoid fever when it was 16 days old, after having been decidedly sick for eleven days. The necropsy was made and the ulcerations of Peyer's plaques were found. The fact that the symptoms in those very small babies are not very urgent and do not appear to be very dangerous, is probably due, to a great extent, to the fact that at that time, at that early age, Peyer's plaques are not very well developed.

At that time we had no Widal, nor anything else that would give us a positive diagnosis; but the diagnosis could be made. There was typhoid in the house, and so on. That is the only case in the newly born that I have seen; but I have heard of a few more cases in babies at a very early age.

CIRCUMCISION IN THE MASTURBATION OF FEMALE INFANTS

ROWLAND G. FREEMAN, M.D.
NEW YORK

The masturbation of female infants while really not common occurs with moderate frequency. At the meeting of this society in 1907, Dr. B. K. Rachford in his presidential address reported fifty-two cases of masturbation in infancy observed by members of this society, only four of which occurred in male infants.

This habit, if neglected, leads to a considerable amount of moral depravity, and on the other hand, it may be fairly controlled by proper treatment. It is, therefore, a serious condition which is fairly controllable and one to which too little attention has been devoted. I say nothing of the masturbation of male infants, for sufficient attention is paid to the hygiene of the male sexual organs so that the sort of irritation which leads to masturbation in infants rarely exists in boy babies. On the other hand, in the female infant there exist fairly regularly marked adhesions between the sensitive clitoris and the surrounding tissues so that on examination the clitoris is frequently found to be buried in these adhesions.

In a normal child these adhesions seem to cause little irritation; but in the nervous, sensitive child they may cause intense irritation and lead to the formation of a habit which, if untreated, may become permanent and exert a most injurious influence over the future development of the child.

The treatment of this condition is both general and local. The general health should be improved by such hygienic measures as are available, and all sources of nervous irritation and excitement should be, as far as possible, removed; but no cure can ordinarily be accomplished by such measures alone. The common use of an apparatus for separating the thighs is of little avail, and the only curative treatment is that applied to the removal of the active source of irritation, the adhesions of the clitoris. These may be separated without the use of an anesthetic. The operation is very painful, and such separation under these conditions is difficult and apt to be followed by the formation of other adhesions. The only satisfactory method of treat-

ing this condition is by circumcision, an operation which should be performed by one who is accustomed to do it, the foreskin being removed as completely as possible.

The result of this operation is most satisfactory. In many cases no recurrence of the habit is noticed after the operation, while in others recurrences will occur; but they are found to be due to adhesions which have formed notwithstanding the cimcumcision.

The existence of so serious a complication in a young infant long before the development of the sexual activity leads one to wonder whether more attention should not be paid to the condition about the clitoris in all young girls with symptoms of nervous irritability. It has been said that the clitoris is an electric button which rings up the whole nervous system. It is my belief that more attention should be paid to the hygiene of this region.

The histories which one obtains of these cases are not always clear. The common history of a child putting its fingers on the genitals at every opportunity, while showing evidence of some irritation, is naturally not sufficient for a diagnosis of masturbation. The real condition is often misunderstood by the parents, but when masturbation exists, a clear history of an orgasm can be obtained after close observation on the part of the attendants.

A few illustrative cases of this sort I will enumerate.

CASE 1.—A female infant aged 10 months was noticed frequently to rub her thighs actively for a certain period and then to become quiet, breaking into a perspiration. This habit had been noticed for several weeks and was becoming more frequent. Examination showed the clitoris to be entirely buried in adhesions. These were somewhat broken down and the clitoris moderately cleared without, however, any effect on the habit, so that an operation was advised. A complete circumcision was done two months ago and at the same time a large adenoid was removed. Since that time no masturbation has been noticed although carefully watched for.

CASE 2.—A girl aged 3 years had probably from the history been masturbating for more than a year, this habit having become worse, especially at bedtime and in the morning. The real character of this habit had only just become evident. The child was circumcised under chloroform anesthesia and at the same time a large adenoid was removed. More than two years have elapsed since this operation without any recurrence of the habit, although a year after the operation the child had an attack of chorea which lasted for about a month.

CASE 3.—A child aged 5 years had weighed only 5 pounds at birth, but had done well until she was 8 months old, when she contracted a colitis followed by the condition known as the infantilism of Herter, from which, however, she recovered again in the course of a year. She is a frail, nervous child, and

it has only recently been noticed that she masturbated. A partial separation of the adhesions without an anesthetic did not remove the habit, so a circumcision was performed under anesthesia, and she has never been known to masturbate since, although five months have elapsed since the operation.

Other cases have shown less favorable results from the operation.

CASE 4.—A child aged 5 years, frail and nervous, with red hair but usually well, was found to masturbate. A circumcision was done under anesthesia followed for a time by a cessation of habit; but three months later the habit was again noticed and on examination new adhesions were found to have formed, and the breaking down of these was followed again by a cessation of the habit for six months. Since that time there have been several periods when the habit was resumed, always associated with adhesions about the clitoris. These, however, have become less frequent and now there is no history of a recurrence during the past year, five years after the operation.

CASE 5.—A child aged 18 months with a cerebral palsy and poor intellectual development gave a history of straining which was supposed to be associated with constipation. She was a premature child weighing 3 pounds at birth who had convulsions on the fourth day. She did not talk or stand, and both the right leg and right arm were weak and of little use. She was said to strain when lying down, would get red in the face, perspire, burrow her head in a pillow and then go to sleep. The clitoris was found to be buried in adhesions and a circumcision was performed. Two months after the operation there were still some slight attempts at masturbation and three months later there were almost none. Five months later it was reported that she had been free from attacks, but for a week past these had recurred, and on examination it was found that new adhesions had formed. She was much better after these were broken down. Six months after the operation she was brought to New York on account of her renewal of the habit, and the clitoris was found fairly buried in adhesions. These were broken down without an anesthetic. This child left four years ago for Australia and it has been impossible since then to keep in close touch with her condition, although the father writes me that she has entirely ceased straining. Here was a difficult case on account not only of the recurrence of the adhesions but also of her mental deficiency.

CASE 6.—Another case very resistant to treatment but always benefited by the breaking down of adhesions was a child I first saw ten years ago who was then 10 months old. She was frail, nervous and had red hair. When only 10 days old what was apparently a typical masturbation was noticed. She was treated with braces, which seemed to control the habit temporarily. She had a suspicious eruption during the first year but never any clear evidence of syphilis. She had a typical orgasm in my office, covering her face with her skirts and rubbing her thighs together, taking a number of deep inspirations and then breaking into a sweat. The clitoris was found bound down by adhesions and a circumcision was performed, after which she was better. Seven months after the operation the mother reported that there had been an entire cessation of the habit until two weeks before. The clitoris was found again buried in adhesions, and a second circumcision was performed under an anesthetic. Since that time there has been a steady gradual improvement in her condition. Occasionally new adhesions have been broken down, but without an anesthetic, and periods of months have elapsed with no attempt at masturbation and there has been no history of the habit now for three years.

CONCLUSION

The masturbation of female infants occurs in nervous children, the stimulating cause being adhesions about the clitoris such as exist in practically all female infants.

The treatment of this condition by braces alone is palliative and not curative. The only curative treatment is by separation of the adhesions and circumcision, and the result of such operative interference is often satisfactory.

211 West Fifty-Seventh Street.

DISCUSSION

DR. JACOBI: I would like to ask Dr. Freeman whether he believes that every case of masturbation in a female infant is caused by these adhesions, or by an affection of the clitoris? I would like to have that question answered, as all his cases are certainly *bona fide,* and they have been cured by the method he recommends. Does Dr. Freeman believe that every case of masturbation is the result of that anatomic anomaly?

DR. FREEMAN: In reply to Dr. Jacobi, I would say that in all these cases that I have seen there have been adhesions of the clitoris, and most of them have been markedly improved by circumcision.

DR. JACOBI: Dr. Freeman has certainly seen a great many cases as discussed. I believe the condition has been met a good many times. I believe it was met in 1874 by myself, when I first wrote on the subject of masturbation in small infants. What Dr. Freeman has told me to-day is a revelation and an addition to my knowledge. Certainly, there must be a nerve irritation about the pudendal nerve and in its neighborhood: and the cases that I have seen, and I believe I have seen a great many cases of masturbation in infants, more in former times than now—are for instance, bad constipation, or fissure of the anus, ever so small; or the presence of oxyurides in the rectum, to which they descend, from the middle part of the colon; or vaginal catarrh, which in some cases is regional, the result of neglect and uncleanliness; in a number of cases the result of an oxyuris traveling from the rectum into that little vagina; or it is the result of a cystitis, which we find not very uncommon, or pyelitis. Pyelitis is recognized now to occur frequently, while some twelve or fifteen years ago it was considered very unusual. Or it is the result of stone in the kidneys, which is by no means rare and causes peripheral irritation. In my experience masturbation is very frequent, and not infrequently during the first year of life. It may last for years.

INFANTILE SCORBUTUS AND THE PASTEURIZATION OF MILK

JOHN LOVETT MORSE, A.M., M.D.
Associate Professor of Pediatrics, Harvard Medical School
BOSTON

In a paper published in the Boston Medical and Surgical Journal, April 2, 1914, I called attention to the rapid and progressive increase in the number of cases of scurvy coming to the Medical Out-Patient Department of the Children's Hospital during the four years ending in 1913, and to the coincident increase in the amount of pasteurized milk used in Boston during this period. This progressive increase, both in the number of cases of scurvy and in the amount of pasteurized milk used, has continued during the first four months of the present year. Table 1 shows these points:

TABLE 1.—Showing Increase of Scurvy

Year	Number of New Cases	Number of Cases of Scurvy	Per Cent.	Percentage of Pasteurized Milk
1904	2,579	3	0.11	
1905	2,533	11	0.43	
1906	2,288	4	0.17	
1907	2.075	7	0.33	
1908	2,407	6	0.24	33.58
1909	2,062	5	0.24	30.68
1910	2,311	11	0.47	63.50
1911	1,967	12	0.61	73.02
1912	1,932	13	0.67	70.59
1913	2,416	21	0.87	80.63
1914 (4 mos.)	723	9	1.24	*85.00

* Estimated.

It was also shown in this paper that a much smaller proportion of the babies afflicted with scurvy in 1913 were fed on the proprietary foods than was the case ten years ago. One of the babies which developed scurvy in 1913 was fed on the breast, and one on condensed milk. In three others there was either no data as to whether milk was used with a proprietary food or, when it was used, whether it was heated or not. In four, a proprietary food was used with milk, the mixture being boiled or pasteurized. The remainder were fed on milk modified in some way. In most instances the mixture was boiled,

in others, scalded or pasteurized. In one, a mixture which was made with pasteurized milk was also boiled.

The food which the babies who developed scurvy in 1914 were taking is shown in Table 2:

TABLE 2.—FOODS ON WHICH INFANTS DEVELOPED SCURVY *

1. Modified milk. Pasteurized at 167° F.
2. Whole milk and farina gruel. Boiled.
3. Whole milk, barley water and milk sugar mixture. Prepared with pasteurized milk.
4. Modified milk. Prepared with pasteurized milk.
5. Allenbury's Food, No. 3, prepared with pasteurized milk. The food was mixed with the milk and boiling water then poured on it.
6. Modified milk with barley water. Prepared with pasteurized milk.
7. Modified milk. Prepared with pasteurized milk, then boiled two minutes.
8. Malted milk and water.
9. Whole milk and oatmeal water, equal parts. The boiling oatmeal gruel was added to the milk.

* The modifications were all reasonable ones.

There can be but little doubt, judging from these figures, that there really has been an increase in the number of cases of scurvy in Boston during recent years. They do not prove, however, that this increase has been due to the introduction of, and progressive use of, the commercial pasteurization of milk. Neither do they prove, striking as they are, that the heating of milk, whether to the temperature of pasteurization or of boiling, has anything to do with the etiology of scurvy. They do suggest, however, that there may be some such connection, and seem to justify further study of the subject along these lines.

70 Bay State Road.

DISCUSSION

DR. GERSTENBERGER: I should like to ask Dr. Morse if he treated and improved the condition by not changing the quality or the composition of the food and by not changing the heat factor, but simply by changing the diet by the addition of orange juice? Just a few weeks ago I saw a case of scurvy develop under my care in an infant that was fed with casein milk. Casein milk, as you know, is heated; and all that I did was to give that child orange juice and keep it on the unchanged casein milk, and the scurvy improved very rapidly.

DR. MILLER: I think that Dr. Morse's contention that scurvy is on the increase, is probably correct. I have in the last two and one-half years seen ten cases of scurvy; all in private practice, some of them in consultation and some in other ways. Some years ago before this society Dr. Northrup spoke about this very matter; and he raised the point that scurvy is not recognized so much now by the average practitioner, because when it was first recognized

in this country, practitioners became very much alive to it, and, if I may so express it, everybody saw scurvy; and then, by improved methods of feeding, it became less and less, until a period was reached in which the practitioners forgot about it and failed to recognize it; a condition now existing. This may be one explanation.

Another reason for this increase may be what I should denominate the bi-pasteurization of milk. In four of my cases the parents pasteurized milk that was already pasteurized, or boiled milk already boiled. This, as I have said, was the only cause I could discover in four of my cases. The increased use of pasteurized milk would, therefore, appear to be an explanation.

I would further call attention to a very interesting fact, namely, that in Stefanson's history of his life in the Arctics for two or three years, he states that he and his companions suffered much from the loss of and absence of fat from their diet. When the fat gave out, that is, when their oil-supply gave out, they would eat to repletion of lean meat, and yet would feel constantly hungry, while two of the party fell ill with scurvy. They sent some of the party back five or six hundred miles to bring a fresh supply of oil, and, when it arrived, their abnormal appetites not only ceased, but the people sick with scurvy got well at once. This would seem to show that scurvy is due to some condition in which the nutrition properties of the diet are not properly balanced.

DR. BLACKADER: I have had somewhat the same experience as Dr. Morse, for whereas, several years ago when we had several cases of scurvy, there was a period in which we had none at all; and gradually, in the last two years, last winter especially, I saw quite a number of cases of scurvy; and, in corroboration of what Dr. Gerstenburger said, in several of the cases that I saw in private practice I made no alteration in the food, but simply added orange juice, with complete relief of the symptoms.

DR. NORTHRUP: In the paper that Dr. Miller refers to, I emphasized the fact that after the commercial pasteurization, the mother or nurse may give the milk a hard boiling; and the second fact is that the new group of practitioners that are growing up, don't know scurvy as the oldtimers did; and I think it is well to emphasize in these meetings the possibilities of scurvy.

DR. JACOBI: Was the increase of scurvy cases absolute, or relative? What was the number of patients ten years ago, five years ago; and what is the number of patients at present?

DR. MORSE: The number of new cases and the total number of cases of scurvy and the percentage were all shown on the chart.

DR. ABT: Holt and Frölich (*Ztschr. f. Hyg. u. Infectionskrankh.*, vol. lxxii) show that pigeons were affected with polyneuritis if fed on cereals for a prolonged period; and also that guinea pigs became scorbutic if they were fed on a cereal diet for any considerable time.

It appears, also, from the observations of the above authors that guinea pigs were peculiarily susceptible to scurvy. Former experiments show that scorbutic changes occurred in dogs which were fed oatmeal and boiled meat. Young hogs acquired scurvy when they were fed rye bread with beef or rice or dried cooked fish. The same sort of experiments were made by Furst (*Ztschr. f. Hyg. u. Infectionskrankh.*, 1912, lxxii, 155). This author fed animals with dried peas and lentils with the result that scurvy occurred. Frölich extended these experiments by feeding guinea pigs raw milk for a prolonged period. He found that the same brittleness of bone which occurred in infantile scurvy was produced, though he observed no effect on teeth or typical hemor-

rhages or changes in the bone marrow. Following this experiment animals were fed boiled milk which had been heated for sixty minutes to 112 C. and scurvy resulted. If the guinea pigs were fed oat meal without the addition of any other food, typical scorbutic symptoms appeared. It is interesting to note, however, that if oat meal plus raw milk were administered, no scorbutic symptoms appeared. Milk which had been heated to 100 C. seemed to possess no antiscorbutic properties, but milk which had been heated to 90 C., fed in addition to the oat meal, showed antiscorbutic properties.

Dr. Joseph Brennemann recently showed a case of scurvy before the Chicago Pediatric Society. An infant 9½ months old that had been fed on a variety of food, though for the most part on Nestlé's food, fell ill with scurvy. On account of the severe diarrhea from which the baby suffered Dr. Brennemann gave the child whole milk, adding two grains of citrate of soda to each ounce of food. The latter, however, was discontinued after the second day. After ten days' treatment with boiled milk the child completely recovered from the scurvy. No orange juice or other commonly termed antiscorbutic food was administered.

The foregoing case brings out the important point that an infant who became scorbutic on a long continued and monotonous diet was favorably influenced by a change in diet, even though the change consisted in the use of boiled milk. Thus it is shown in the preceding case that even boiled milk was antiscorbutic.

DR. CRANDALL: I can not help but think that a part of this increase is due to the fact that the present generation of practitioners is not quite as alive to the disease as they were twenty years ago. It was just twenty years ago, at one of these meetings, that Dr. Northrup and I presented a paper on this subject. Up to that time only an occasional case had been reported. It was very shortly after that, that this society conducted a collective investigation. For a time scorbutus was a very popular subject for medical writing. These subjects, I have observed, go in cycles. I write each year a review of pediatric literature for publication, and have been surprised to see how certain diseases are considered of certain periods. There were, for instance, a number of years when more was written on poliomyelitis and cerebrospinal meningitis than on anything else. I have been surprised for a number of years to see how little has been said about scurvy. I think a good many of the younger general practitioners are not awake to it, and do not recognize it as promptly as they did fifteen or twenty years ago. That may be one reason for its appearing in the dispensaries and hospitals as Dr. Morse reports.

DR. FREEMAN: Concerning the relationship of scurvy to pasteurized milk, I would say that in New York during the past year almost all the milk has been commercially pasteurized between 140 and 145 F. for twenty minutes by order of the health department. If pasteurized milk causes scurvy one would expect a large increase in the number of cases in our clinics due to this regulation, but so far as I remember, in our very large clinic at the University and Bellevue Hospital Medical Schools, there has not been a single case of scurvy during the past winter.

DR. SOUTHWORTH: There is one offsetting factor; I am not sure whether it was mentioned. It is true that there is more pasteurized milk used than formerly, but at the same time, the practice of giving young children orange juice or some fresh principle has increased tremendously under the pediatric teaching of the last twenty years. This opens the interesting question whether

we should or should not have a much larger amount of scurvy if young children were not so generally given orange juice, even in the fairly early months of their life.

DR. PISEK: I would like to confirm what Dr. Freeman said about the absence of scurvy in New York. In looking over the abstract of Dr. Morse's paper it occurred to me that I had not seen a case within the past year. And I wondered if this was not so because of the process of pasteurization of our milk which is now mainly the Holding process. In other words, commercial pasteurization is being done in the modern way and scurvy is rarely seen. Then I should like to ask Dr. Morse, for purposes of record, about the ages, and whether these children had orange juice or any other food except those that he indicated?

DR. HEIMAN: I would like to confirm what Dr. Freeman and Dr. Pisek said. I see a great many children at our clinics; and this year I have not seen a case of scurvy. I thought it was the general impression among pediatrists that it was not the pasteurization of milk, but the artificial foods that were responsible for scurvy. We have also seen rickets following pasteurized milk and we have seen equally as much rickets following non-boiled milk; and I believe that is entirely due to cows' milk itself, and not to the pasteurization. Scurvy is probably on the decrease, at least in New York, as far as we can judge from our clinics.

DR. GRIFFITH: I cannot resist the temptation of calling Dr. Morse's attention again to our collective investigations. Circumstances made me perhaps more familiar than anyone else with the figures, inasmuch as, in the capacity of chairman of the committee, I had to spend very many late hours in the analysis of them. I do not think that we have got very much further than we had at that time. The conclusions of the committee, if Dr. Morse will remember, were that there was something wrong with the food; but we did not know exactly what. Now, if you go over those figures, you will find that a change of some sort seemed to be the thing required. There were ten of these children, if I remember correctly, who were breast-fed; and there was certainly nothing which could be attributed to pasteurization or sterilization in these cases. There were cases, too, in which the mere change from sterilized raw milk did good; others in which the change from raw milk to a heated milk did good. There seemed indeed to be something wrong which varied in every instance with the individual child. There must, of course, be some scientific basis, something on which one can finally put one's finger, but I do not think we have yet determined what this is; and we are not in the position to blame pasteurization, sterilization, or rawness of milk.

DR. MORSE: In the first place, as to the foods that these babies were taking, I gave that in my paper; and these babies were not getting orange juice or anything fresh. There is one very noticeable thing which I did not bring out here in detail, because it appears in the other paper, and that is, that ten years ago most of the babies that had scurvy were on proprietary foods, while last year most of the babies that had scurvy were on modified milks. So far as I can see by looking over these modifications, they were reasonably good ones. That is, proprietary foods have not played the part that they used to and the only constant factor in this series has been this increased pasteurization of milk.

DR. HAMILL: You emphasized in your paper the fact that it was commercially pasteurized milk, as I understood you; did you not?

DR. MORSE: There is an increase in the general use of pasteurized milk; and there is also this added fact of pasturization and boiling in the home; but the cases I reported this morning occurred in 1914; a majority of them occurred after the use of commercially pasteurized milk, which was not pasteurized at home afterwards.

DR. HAMILL: Do you know the process by which the milk was pasteurized commercially and the temperature used? It is extremely important to state explicitly the method of pasteurization and its details whenever the process of commercial pasteurization is referred to, since the results depend on the method chosen and the proper carrying out of its technic. Certain methods, if properly carried out, will render milk safe, from the point of view of infection and without in any way affecting nutritive value.

DR. MORSE: At present, in Boston, the regulations are not definite enough as to what pasteurization is. My own belief is that this milk was pasteurized in various different ways commercially, and probably that a certain amount of what in Boston is called pasteurized milk really is not pasteurized milk; that is, our regulations are not as good as they ought to be.

As to the question of giving these babies orange juice in the treatment. They were, of course, all given orange juice at once. It doesn't seem to me justifiable to do anything else, since we know that it will cure them right away. We give them orange juice anyway. Sometimes we change the food and sometimes we don't. I have, of course, in some instance allowed the baby to continue to take the foods, stopping the heating of the milk, when the baby was thriving in other ways; but it seems to me that we ought to cure them as quickly as we can with the orange juice.

As to the failure to recognize the disease, it would seem to me that certainly at Boston the physicians ought to know more about scurvy now than they did a number of years ago, because it has constantly been talked of and written about there, and certainly nobody goes through the Harvard Medical School that doesn't know scurvy. A great deal of attention is paid to it.

I can not understand the difference in the relative frequency of scurvy in Boston and New York. Certainly, the cases that we got were scurvy; and I have every reason to believe that the gentlemen here from New York know scurvy when they see it.

I didn't mean in my paper to make any claim that the heating of milk is the cause of scurvy. I carefully said, "a possible explanation for it"; because, in looking over the literature in relation to the effects of the pasteurization of milk on babies, as I have had to twice in the last few years, I had to conclude that there was nothing in the evidence, either clinically or experimentally, which showed that the pasteurization of milk had any effect on the nutrition of babies, or in the causation of either scurvy or rickets. Yet here we meet these figures. They merely seem to me to be suggstive that perhaps there is something in it which we have not been able to explain theoretically or experimentally. I feel as Dr. Griffith does: that we don't know the cause of scurvy and that all we do know is that there is something wrong with the food; and it is for the future to determine what that is.

EXPERIENCES WITH THE HIGH CALORIC DIET IN TYPHOID FEVER OF INFANTS AND YOUNG CHILDREN *

L. E. La FETRA, M.D.
Visiting Pediatrist, Bellevue Hospital

AND

LOUIS C. SCHROEDER, M.D.
Resident Physician, Pediatrics Service, Bellevue Hospital

NEW YORK

All pediatrists are familiar with the general course, symptoms and complications of typhoid fever in infants and children when the diet has been limited in accordance with the older ideas of the effect of fever on the digestive capacity. In this society studies of typhoid have been recently reported by Drs. Hand and Gittings, by Drs. Koplik and Heiman and by Dr. Griffith.

One of the main features of typhoid fever has always been the marked emaciation, and closely related to the great loss in weight were the lowered resistance to complications and to relapses and the tendency to toxemic nervous symptoms.

The loss of weight in typhoid, as in starvation and in other fevers obeys the general law that the carbohydrate glycogen stored in the body is at first utilized, and after this the fat and the protein. A characteristic feature of the metabolism of prolonged high fever is the marked loss of body nitrogen.

The loss of weight in typhoid has been attributed to (1) partial starvation, (2) the febrile temperature, and (3) the toxic destruction of protein.

Partial starvation has always been recognized, and the effort has been made by most clinicians to prevent the loss in weight by increasing the diet.

Febrile temperature or artificial heat increases protein metabolism, and probably also the metabolism of fat and carbohydrate. To have such an effect the fever must be high and prolonged. The infectious fevers cause a greater loss of nitrogen than starvation or artificial

* From the Pediatric Service, First Medical Division, Bellevue Hospital, New York.

temperatures, so that it has been assumed that the bacterial toxins exert a destructive influence on the body cells. It has been proved by Coleman and his co-workers that in adults it is possible by the high caloric diet in typhoid to prevent not only the loss of nitrogen, but the loss of weight as well. Weight equilibrium can be maintained in typhoid patients by a sufficient diet.

The main objections to such a diet are: (1) the patients cannot digest and absorb so much food; (2) the amount of fat will cause alimentary disturbance and acidosis; (3) granted absorption, patients do not require the amount of food advocated.

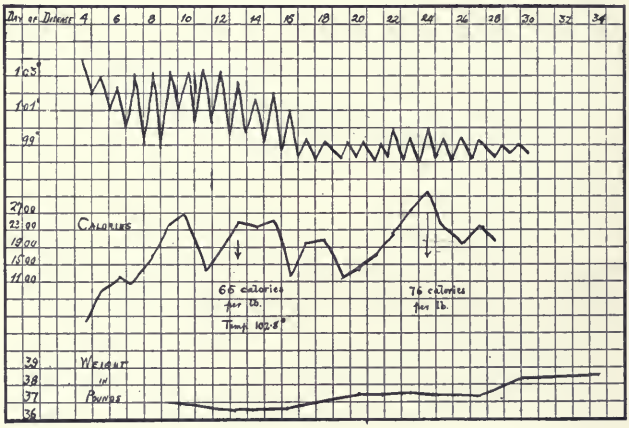

Fig. 1.—High caloric diet in febrile period.

Du Bois has shown that the typhoid patient has a remarkable capacity to absorb large amounts of food. He concludes from his metabolism studies that typhoid patients, throughout the disease, can absorb carbohydrate and protein as well as normal individuals; they can absorb also very large amounts of fat; but the percentage of absorption is somewhat lower than the normal, especially in the early part of the disease.

Coleman states that in his 111 cases the increased amount of fat seldom caused any diarrhea, and there was no clinical evidence of acidosis; moreover, no acetone bodies appeared in the urine and the ammonia nitrogen in the urine never exceeded 2 gm., being usually below 1 gm.

As to the food required, it was found clinically that patients lost weight when an excess of food was not given; although an excess of from 1,000 to 2,000 calories were furnished over what the patient expended, there was a loss both of weight and of nitrogen unless such excess was given. In the later stages of the disease the excess is utilized for storing fat.

From the bacteriologic point of view Kendall's[1] studies are important in this connection. He says:

Physiologists have long known that carbohydrate spares body nitrogen, therefore physiological loss of weight due to simple starvation should be largely prevented by such foods. In Coleman's cases not only was the loss of weight

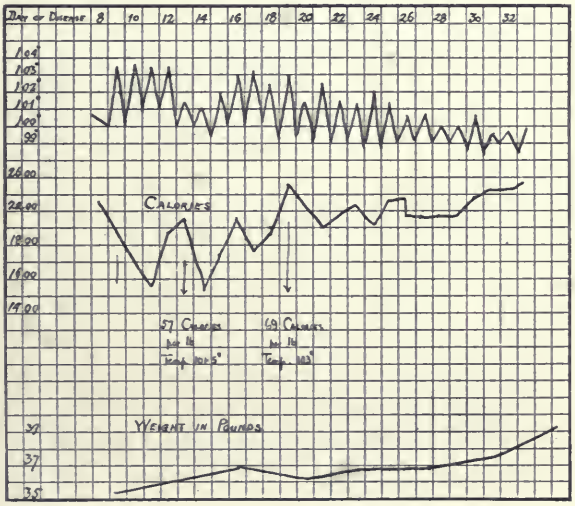

Fig. 2.—High caloric diet in febrile period.

attributable to partial starvation prevented, but in addition, practically all the loss attributable to the febrile reaction and to toxemia was markedly reduced. The general symptoms of toxemia were greatly modified.

Kendall further says:

The explanation that presents itself to account for this reduction in toxemia when the patient is on a liberal diet containing an excess of carbohydrates is a simple one. The maintenance of a high physiological sugar content in the body, and consequently in the blood, influences the metabolism of the typhoid bacilli, directly limiting their action on the body tissues. The typhoid bacilli

1. Kendall, Arthur I.: Boston Med. and Surg. Jour., June 5, 1913, p. 825.

get their *fuel energy* largely from sugar (dextrose), although their nitrogen requirements are necessarily of protein origin. The waste from the utilization of carbohydrate may be irritative, but is practically never toxic. It is almost certain that the waste products of their *structural* requirements are of comparatively little magnitude. The bacterial waste from fuel needs, particularly when this fuel waste is derived from protein break-down (body tissue in this case) is the more dangerous factor.

It is comparatively simple to understand the changes in the metabolism of the bacteria in the intestinal tract where they are directly in the path of the carbohydrate that is added to the diet and in this connection, the change in the nature of metabolism of the normal endogenous intestinal flora may well play

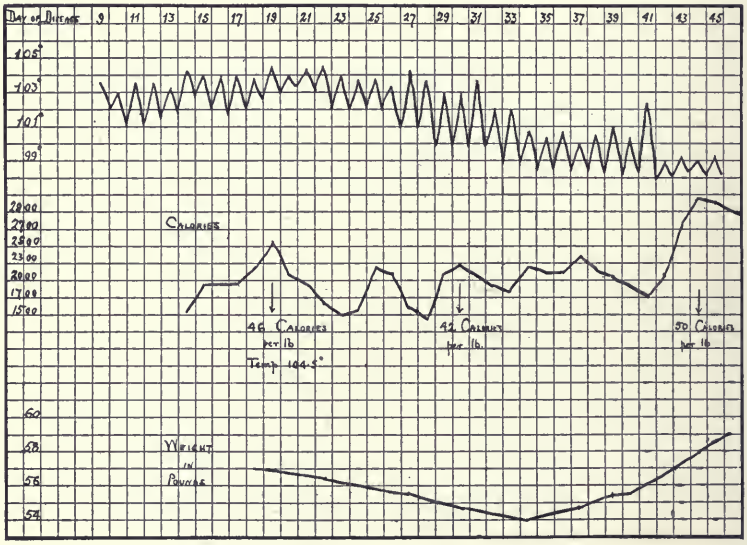

Fig. 3.—High caloric diet in febrile period.

a part. Bacillus coli, for example, forms lactic acid in place of indol, if sugars are available. This may lighten the load on the liver and kidneys, since practically no indol is formed to be excreted through the urinary tract. In the body itself, on the contrary, the typhoid bacilli might at first sight, seem to be widely separated from utilizable carbohydrate. Automatically, typhoid bacilli cause focal necrosis in the various organs of the body, but it should be remembered that necrotic tissue is readily permeable to crystalloids. It is very probable that the dextrose of the blood, which is maintained at its highest physiological level on a diet rich in carbohydrate (carbohydrate absorption is only slightly impaired in typhoid) seeps into the areas where the organisms are active and therefore reaches them without difficulty. This dextrose is utilized by the typhoid bacilli in preference to the tissue of their host for their fuel. The character of the products of fuel wastes consequently becomes *acid* in place of

being alkaline, nitrogenous and toxic, as would be the case of the dextrose content of the blood were diminished. This explanation involves no theoretical difficulties, and the observed clinical results of a liberal carbohydrate diet in reducing the loss of body protein (loss of weight) in suppressing the toxic state, and in general favorably influencing the course of the disease are in accord with the chemical and clinical results observed empirically in many cases where this diet has been introduced. . . . A mortality of 12 among 174 cases of typhoid treated along these lines by Coleman, Croker and Gardner shows what can be done.

Small feedings frequently repeated are important in treating typhoid fever from the bacteriological point of view, so as to maintain a maximal concentra-

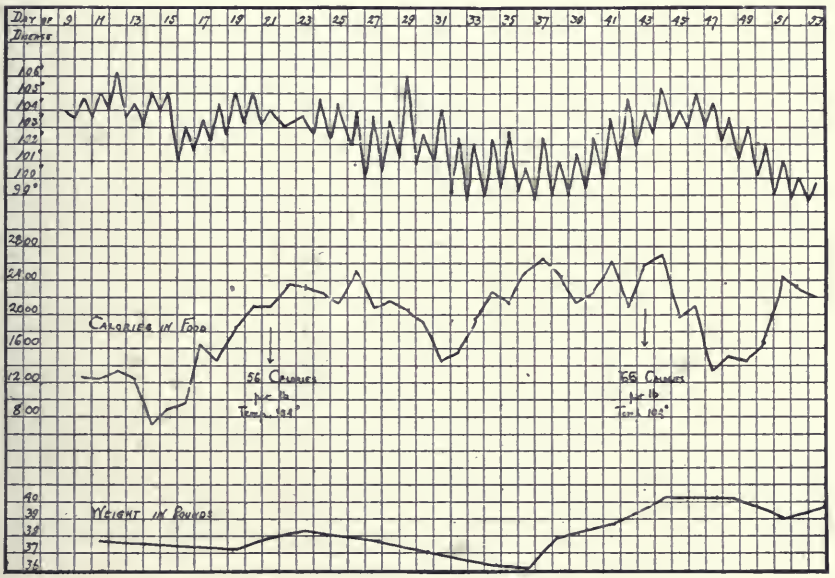

Fig. 4.—High caloric diet. Case with recrudescence.

tion of utilizable sugar, in the intestinal tract and in the body as well. Infrequent feedings fail to do this because the carbohydrate is as a rule rapidly absorbed from the intestinal tract, leaving a residue of protein if the bowel movements are not frequent. The abstraction of the carbohydrate forces the bacteria to utilize protein, be it body tissue or intestinal residuum.

The object of a diet rich in carbohydrate is two fold: *Physiologically* to provide the patient with a readily assimilable food requiring a minimal amount of digestive energy to prepare it for the tissue needs; and *bacteriologically* to shift bacterial metabolism from the destruction of body tissue for their food requirements to the utilization of carbohydrate for at least the major part of their dietary needs. This does not result in annihilation of the invading bacteria, but it certainly approaches their metabolic reformation.

From the point of view of the clinical pathologist, Dr. J. C. Torrey[2] in a recent communication to the New York Academy of Medicine, stated that the high carbohydrate diet encourages the growth of the acidophils; that on a protein diet, such as milk, the putrefactive type predominated; both he and Dr. Charles Norris, the pathologist of Bellevue Hospital, showed that when acidophil bacteria were present in abundance, it was extremely difficult to recover typhoid bacilli from the

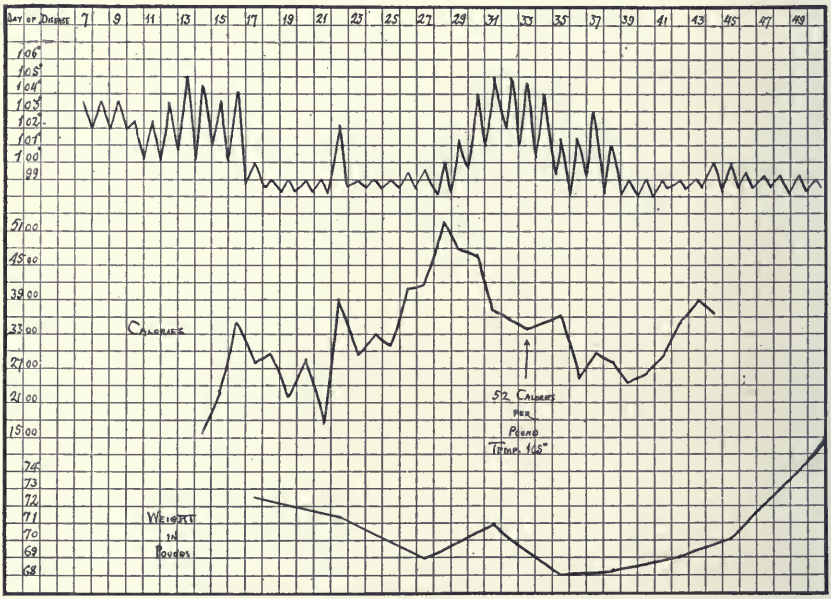

Fig. 5.—High caloric diet in typhoid. Case with relapse.

stools. High fat diet temporarily restrained the growth of acidophils; but when the carbohydrate was resumed, the acidophils grew rapidly again.

The metabolism studies of Coleman and Schaeffer and of Du Bois, and the clinical observations of Coleman and his coworkers at Bellevue Hospital, have shown that in adults a much more liberal diet than formerly used is advisable. It was because of interest aroused by this

2. Dr. Torrey's studies have been published under the title, "The Fecal Flora of Typhoid Fever, and Its Reaction to Various Diets," in The Journal of Infectious Diseases, 1915, xvi, 72.

work that we have been anxious to test the value of the high caloric feeding in the typhoid fever of children.

It is seldom that one has the opportunity in New York to see more than an occasional sporadic case of typhoid in children, so that when a considerable epidemic of typhoid occurred in one part of New York, the opportunity was afforded to make a thorough trial of Dr. Coleman's plan of feeding. Some results of our experiences with this diet seem

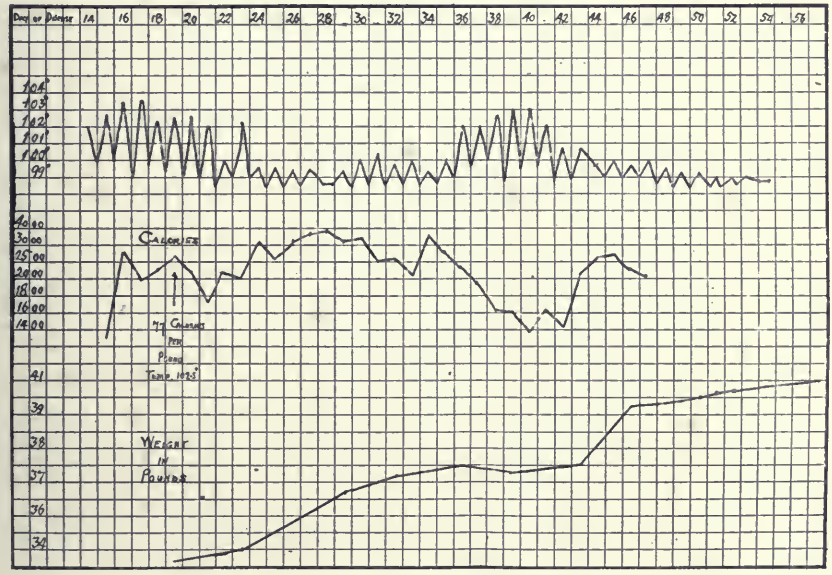

Fig. 6.—High caloric diet in typhoid. Case with relapse.

worthy of record. For aid and counsel and for valuable suggestions throughout the epidemic, as well as permission to make free quotations from his writings, we wish to thank Dr. Coleman most heartily.

TREATMENT

The routine treatment was sponge baths for temperature of 103.5 F. The patients were kept on the balconies or in rooms with open windows.

Incidentally it may be stated that typhoid vaccine was administered in seven cases, from one to six doses being used, starting with 50 million and increasing by 50 million each successive dose. There was no perceptible influence on the course of the disease.

GENERAL DIET

The children were fed at three-hour intervals, beginning at 6 a. m. and continuing until 9 p. m., six feedings in the twenty-four hours. The articles given were such as would be prescribed for a child in health, with the exception that the only meat used was creamed chicken. In order to increase the caloric value of the liquid food, which was practically always milk, lactose and cocoa and usually cream were added. In no case was an exclusive milk diet used; every patient took cereals in some form. Eggs and toast were offered from the beginning, and it is largely a matter of the nurse's tact and encouragement how soon a diet high in calories will be taken. At first only a part of the food offered will be taken, and the patient's likes and dislikes must be respected and taken advantage of.

Aside from giving a total amount of food with sufficient caloric value, there must be a certain minimum of protein in the diet. Voit's standard for adults was something over 100 gm. per day, but more recent work has shown that even less would do, but that some 70 gm. should be afforded. Inasmuch as no metabolic studies on children suffering from typhoid have been made, we know of no standard for the daily protein ration in childhood. In our series of cases no attempt was made to have either an upper or a lower limit of protein, but the general rule was adhered to, to have the protein calories not less than 7 nor more than 15 per cent. of the total number of calories given.

Some typical meals of children on the high caloric diet with quantity of food taken and number of calories furnished are given herewith.

TYPICAL DIETS

Angelo R., on the forty-ninth day of disease received 2,214 calories:

6 a. m.: 4 ounces milk, 2 ounces cream, 2 eggs, 2 slices bread.
9 a. m.: 5 ounces milk, 2 ounces cream, 3 crackers.
12 m.: 3 ounces potatoes, 1 egg, 1 slice bread, 6 ounces milk, 2 ounces cream, 2 drams milk sugar.
3 p. m.: 1 ounce ice cream.
6 p. m.: 1 ounce apple sauce, 4 ounces milk, 2 ounces cream, 1 slice bread.
9 p. m.: 4 ounces milk, 2 ounces cream, 2 drams milk sugar.

Leonard B., on November 9 received 2,410 calories:

3 and 6 a. m.: 2 eggnogs with eggs, ½ ounce milk sugar, 2 ounces cream, 4 ounces milk.
6 a.m.: 4 ounces milk.
9 a. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg (eggnog), ½ cracker.
12 m.: Eggnog, 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg.
3 p. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg.

6 p. m.: Cocoa, 490 c.c.
9 p. m.: Eggnog with 4 ounces milk, 2 ounces cream, 2 eggs, ½ ounce milk sugar.

Leonard B., on December 10. during convalescence, received 7,206 calories:
6 a. m.: 10 ounces cereal, 2 eggs, 4 ounces milk, 2 ounces cream, 4 slices bread.
9 a. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 4 crackers.
12 m.: 19 ounces potatoes, 6 ounces cream, 4 ounces custard, 8 slices bread.
3 p. m.: 6 ounces milk, 2 ounces cream, 3 crackers.

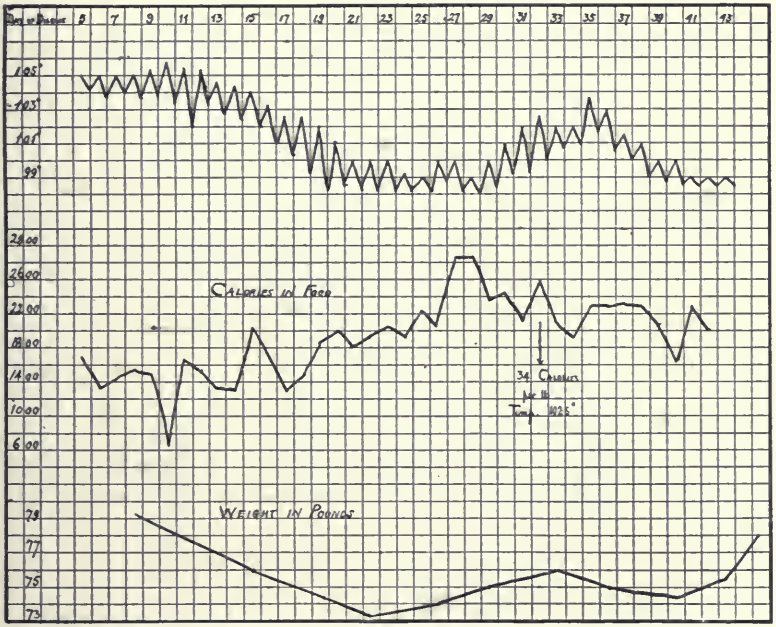

Fig. 7.—High caloric diet. Case with short relapse.

6 p. m.: 1 egg, 2 ounces apple sauce, 4 slices bread, 4 ounces milk, 2 ounces cream.
9 p. m.: 4 ounces milk, 2 ounces cream, 3 crackers.

J. Corcoran, on the ninth day of disease with temperature ranging from 100 to 103 F., received 2,430 calories:
6 a. m.: 4 ounces milk, 2 ounces cream, ½ slice bread, 1 egg.
9 a. m.: Cocoa, 490 c.c.
12 m.: 4 ounces milk, 2 ounces cream, 1 ounce milk sugar, 1 egg, 1 slice bread.
3 p. m.: 4 ounces milk, 2 ounces cream.
6 p. m.: 4 ounces milk, 2 ounces cream, 1 ounce milk sugar, 1 egg, 1 slice bread.
9 p. m.: 4 ounces milk, 2 ounces cream.

The calculations of the caloric values were made from the following list prepared by Dr. Coleman:

FOOD VALUES — SOLIDS

Food	Amount	Calories
Apple sauce	1 ounce	30
Bread	Average slice (33 gm.)	80
Butter	1 pat (⅓ ounce)	80
Cereal	1 heaping tablespoonful (1½ oz.)	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Custard	4 ounces	250
Cream chicken	1 ounce	50
Egg	1 (2 ounces)	80
Egg (white)	1	30
Egg (yolk)	1	50
Lactose	1 tablespoonful (9 gm.)	36
Milk (whole)	(1 pint 350 gm.) 1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane	1 lump	16
Sugar, milk	1 tablespoonful (9 gm.)	36
Toast	Average slice	80

For practical purposes, the milk sugar may be measured in a medicine glass.

Each measured ounce equals 18 gm. in weight. If milk sugar is added to water in the proportion of 24 gm. to 30 c.c., and the water brought to the boiling point, the milk sugar is completely dissolved. Such a solution made daily, or just before use, will be found convenient in administering the diet.

FOOD VALUES — LIQUIDS

Cocoa with milk:	Calories
1 rounding teaspoonful of cocoa	50
2 ounces of milk sugar	240
4 ounces of milk	80
2 ounces of cream	120
	490

Mix the sugar and cocoa; cook in the milk until dissolved. Serve with cream.

Cocoa with cream:	Calories
1 heaping teaspoonful of cocoa	50
2 ounces of milk sugar	240
½ cup of water	...
3 ounces of cream	180
	470

Mix the cocoa and sugar, add the water and boil. Then add the cream, or use less cream and serve with whipped cream.

Coffee:	Calories
1½ ounces of milk sugar	180
4–5 ounces of strong coffee	...
2 ounces of cream	120
	300

Lemonade:	Calories
4 ounces of milk sugar	480
7 ounces of cold water	...
2 tablespoonfuls of lemon juice (or to taste)	...

Boil the sugar and water for two minutes, add lemon juice to taste, strain and cool.

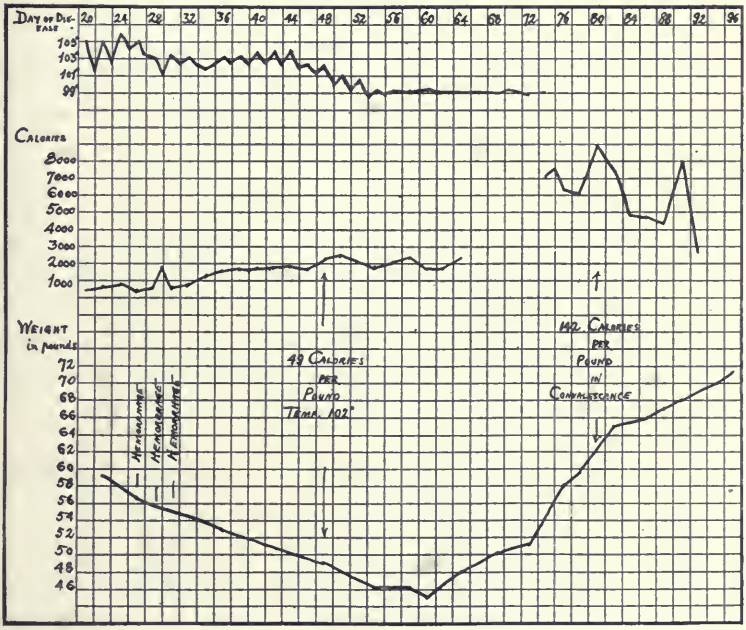

Fig. 8.—Severe case with hemorrhages. Shows loss of weight in spite of liberal feeding during febrile period, but rapid gain in convalescence while taking food of very high caloric value.

The number of calories that were actually taken after the first few days, and that seemed from a clinical point of view to have been satisfactorily utilized, varied from 100 to 300 calories per kilogram. As regards utilization of the food the urine in these cases very seldom showed any more than a slight trace of indican.

It was at first thought that a high range of temperature would in itself interfere with the taking of so much food; this was found to be

TABLE 1.—GENERAL HISTORY OF PATIENTS IN CASES 1 to 43

Case No.	Name	Age, Years	Prodromal Period, Days	Time in Hospital,* Days	Chief Complaint	Nose Bleed	Rose Spots	Spleen Enlargement	Bowels
1	S. Silverman	1 1/12	3	32	Fever	None	++	None	Diarrhea.
2	Freda Gerke	2	10	28	Fever	None	++ Appeared in 4 days	None	Regular
3	G. Geiche	3½	7	23	Headache; tire; fever; pain in stomach	None	++++	+	Loose.
4	A. Dunn	4	2	27	Headache; pain in abdomen	None	++	++	No diarrhea.
5	Margaret Kronek	4	7	14	Fever; headache	?	None	None	Costive.
6	N. Woehinsky	4	7	20	Abdominal pain; headache	None	++	++	No diarrhea.
7	W. Guthart	4½	13	16	Malaise	Yes	++	++	Costive.
8	Sadie Penna	4½	14	26	Abdominal pain; fever	None	++	++	Costive.
9	H. Autmajor	5	3†	4	Fever	None	?	None	Costive.
10	Joseph Cochrane	5	7	28	General malaise	None	None	?	No diarrhea.
11	Catherine Shea	5	6	19	Headache; fever	None	None	+	Costive.
12	M. Geiche	5	5‡	25	Headache; fever; pain in stomach	Negative	+++	+	Loose.
13	R. Zingales	5	5	31	Malaise; headache; fever	None	++	None	None.
14	Rosee Zingales	5	4	21	Headache; fever	None	?	None	Slight diarrhea.
15	George Zoul	5	8	22	"Pain in belly"	None	+	None	Diarrhea.
16	Rocco Gariballi	6	3	18	Fever; headache	None	++	None	Slight diarrhea.
17	Aaron Psau	6	8	16	Fever; headache	?	None	+++	Costive.
18	E. Corcoran	6	7	19	Pain in right side	None	None	None	No diarrhea.
19	Jas. Sloan	6	3¶	43	Fever; occipital; sudden onset; three days	?	None	++	Costive.
20	Catherine Dorso	6½	4	14	Fever	None	++	+++	Costive.
21	John Kellcper	7	8 In bed 2	47	Fever; sleepiness; pain in stomach	?	None	++	Diarrhea for one day.
22	Mike Forlenza	7	14	28	Headache	None	Few ?	None	Constipated.
23	Mary Maucum	8	9	52	Fever; headache	None	++	++	Constipated.
24	Thos. O'Brien	8	7 Bed at once	15	Headache; fever	+ 5 days after adm.	? None	None	Constipated.
25	Coro Guselau	8	5	17	Headache; pain in stomach	?	+++	++	?
26	Olga Medvill	8	3	23	Headache; fever	None	+++	++	More open than usual.
27	Mary Grossman	8	14	44	Headache	None	+++	++	Costive.
28	Julie Schluppe	8	10	28	Headache	Slight	++	++	Loose.
29	Josephine Zanger	9	30	30	Fever	+	++	?	Costive.
30	Josie Campioni	9	7	27	Vomiting; headache	?	None	None	Constipated.
31	Mary Piazza	9	5	34	Fever; headache	None	++	++	None.
32	Angelo Rone	9	10§	..	Fever	None	++	++	?
33	Abe Psau	9	10	16	Headache; fever	None	?	+	Costive.
34	John Purcell	9	10	23	Fever; loss of appetite	None	++	+	Diarrhea.
35	A. Damprosio	10	7	13	Fever; pain in abdomen; headache	Negative	+++	+	Costive.
36	Mary McKenna	10	6	17	Fever; apathetic	None	+++	++	No diarrhea.
37	W. Medville	10	7	31	Chilliness; fever	None	++	++	Costive.
38	Angelina Gariballi	10	5	22	Fever; headache	None	++	++	No diarrhea.
39	C. McCarthy	10¾	7	26	Tire; fever; pain in neck for two days but no headache	Yes	+	+	Costive.
40	F. Windberg	11	6	43	Loss of strength and appetite; fever and slight cough	None	?	+	No diarrhea.
41	Sophie Girdansky	11	3	44	Headache	Slight on day of admiss.	++	None Later ?	Constipated.
42	Rose Newman	11	7	42	Headache; general malaise	None	++	++	
43	Leonard Bisniginns	11	19‖	79	Fever	None	++	Not felt	No diarrhea.

* There is some question as to the length of convalescence.
† Cough 1½ months. ‡ Confined to bed at once. ¶ Sudden onset. § Three weeks before admission. ‖ Sick before admission.

TABLE 2.—Tests Made and Complications Seen in Cases 1 to 43

Case No. and Distention	Hemorrhages	Blood Counts	Widal and Blood Culture	Urine	Other Complications
1—None	None	9/7: 11,500 W. C.; P. 42; Ly., 58. 9/10: 17,500 W. C.; P., 57; Ly., 33; L. mono., 6; Trans., 2; Baso., 2.	9/10: W., paratyphoid, pos.	9/7: Albumin light cloud. Sugar, 0.	Both great toes and fourth and fifth fingers of left hand have pus. Double otitis. Gastro-enteritis. Pus shows B. paratyphoid.
2—None; held tense.	None	9/25: 12,500 W. C.; P., 50; Ly., 50.	9/23: W. pos.; 9/27: B. c., pos.	None	No vomiting.
3—None	None	9/13: 17,000 W. C.; P., 60; Ly., 40. 9/22: 10,000 W. C.; P., 34; Ly., 61.	9/18: W., pos. 9/20: B. c., pos.		Refused part of feeding on first four days. Not afterward. Vomited, 16, 17, 19, 21.
4—Slight for two weeks.	None	9/22: 15,500 W. C.; P., 55; Ly., 45.	9/18: W., pos. 9/20: B. c., pos.	9/23: Ft. trace alb. Sugar, 0. Few leukocytes.	Slept fairly well during vomiting period. No mental disturbance.
5—None	None		10/3/13: Widal neg.		None.
6—Slight on admission and for ten days; then soft.	None. Streak of blood on Octoer 3.	9/16: 12,200 W. C.; Ly., 76; P., 15; L. mono., 9.	9/20: W. pos. 9/22: B. c., pos.	9/23: Sugar, 0. Albumin, 0.	None.
7—None	None	10/14: 9,200 W. C.; P., 43; Ly., 57.	10/13: W. pos. 10/18: B. c., neg.	None	None.
8—None	None	9/26: 5,000 W. C.; P., 58; Ly., 42.	9/27: W. pos. 10/1: B. c., pos.	None	None.
9—None	None	10/9: 6,200 W. C.; P., 50; Ly., 50.	10/9: W., pos.		Bronchitis? Measles. By Roentgen-ray miliary nodules found.
10—None	None	9/30: 10,000 W. C.; P., 66; Ly., 34.	10/2: W., pos. 10/4: B. c., neg.	None	Stye on left eye. Otherwise none.
11—None	None	10/9: 13,400 W. C.; Ly., 20; P., 80.	4/10: W. pos. 6/10: B. c., neg.	None	Vomited three days. October 3, 4, 6.
12—Slight, on admission full through course. Never rigid.	Slight trace of blood on December 2. No change of diet.	9/7: 23,000 W. C.; P., 66; Ly., 34. 9/16: 15,500 W. C.; P., 60; Ly., 59; Baso., 1.	9/18: W., pos. 9/20: B. c., neg.	9/20: Albumin, 0. Sugar, 0. Modcrate No. of leukocytes.	Vomited twice. Otherwise none.
13—None	None	18,000 W. C.; P., 67; Ly., 33.	9/23: W., pos. 9/27: B. c., pos.	None	Ecchymosis over left eye. None.
14—None	None	10/28: 9,500 W. C.; P., 56; Ly., 44.	10/29: W., pos. 10:29: B. c., pos.	1/11, 1/12, 1/14: Albumin, neg. Casts, neg. Sugar, neg. Ind., neg.	Readmission; appetite poor until October 9. Then very good.
15—9/19, none; 9/20, slight; 9/25, none.	None	9/19: 23,400 W. C.; P., 77; Ly., 15; L. mono., 8. 9/23: 11,500 W. C.; P., 63; Ly., 37.	9/22: W., pos. 9/25: B. c., neg.	9/24: Albumin, faint trace. Sugar, 0. Casts, 0.	Few crackling râles noted September 20 and September 22 at left apex and right base. Cleared up before discharge.
16—None	None	10/15: 4,000 W. C.; P., 71. Ly., 29.	10/16: W., pos. 10/18: B. c., pos.	None	No nervous symptoms at any time.
17—None	None	9/20: 6,600 W. C.; P., 56; Ly., 42; L. mono., 2.	9/22: W., pos.; 9/25: B. c., neg.	Albumin, trace. Sugar, 0. Few hyaline casts.	
18—None	None	10/9: 6,000 W. C.; P., 75; Ly., 25.	10/10: W., neg. 10/14: B. c., pos.	None	None.

TABLE 2.—TESTS MADE AND COMPLICATIONS SEEN IN CASES 1 TO 43—(*Continued*)

Case No. and Distention	Hemorrhages	Blood Counts	Widal and Blood Culture	Urine	Other Complications
19—None	Slight trace of blood.	8/26: 12,500 W. C.; P., 72; Ly., 28. 8/27: 7,800 W. C.; P., 63; Ly., 30. 9/12: 17,000 W. C.; P., 54; Ly., 46; L. mono., 5.3; Trans., 1.7.	8/17: W., neg. 9/3: W., pos. 9/11: B. c., pos.	8/25: Faint trace albumin. Mammoth puscells. 8/26: Albumin, trace. (Hyaline and granular casts many.)	Tenderness and resistance in right upper quadrant.
20—None	None	9/27: 9,500 W. C.; P., 55; Ly., 45.	10/2: W., pos. 10/4: B. c., neg.		None. Uneventful recovery.
21—Slight on admission; none after.	None	9/5: 7,400 W. C.; P., 34; Ly., 54; L. mono., 10; Eo., 2. 8/26: 9,100 W. C.; P., 75; Ly., 13; L. mono., 8; Trans., 4.	8/27: W., neg. 9/27: B. c., pos.	8/26 and 9/24: No sugar. No albumin.	None.
22—Tense for first six days. Then soft.	None	10/29: 10,000 W. C.; P., 57; Ly., 43.	10/29: W., pos. 11/1: B. c., neg.	11/11 and 11/12: Albumin, neg. Sugar, neg. Ind., neg. Casts, neg.	Acute bronchitis for from four to five days.
23—On admission and for sixteen days.	None	9/18: 7,000 W. C.; Ly., 66; P., 34. 9/22: 7,000 W. C.; Ly., 60; P., 40. 10/15: 7,500 W. C.; P., 54; Ly., 46.	9/10: W., pos. 9/14: B. c., pos.	None	Delirious for first week. Boils, parotitis, bronchopneumonia, double otitis media.
24—None	None	9/24: 7,500 W. C.; P., 43; Ly., 57.	9/22: W., neg. 9/24: W., neg. 9/25: B. c., neg.	Albumin, neg. Sugar, neg.	None.
25—None	None	9/18: 5,200 W. C.; P., 54; Ly., 36; L. mono., 10.	9/22: W., pos. 9/22: B. c., neg.	Albumin, 0. Sugar, 0.	None.
26—None	None	9/9: 10,500 W. C.; P., 60; Ly., 40.	9/14: W., pos. 9/16: B. c., pos.	Sugar, 0. Albumin, 0.	None.
27—Slight; none after relapse.	None	None	9/13: W., pos. 9/14: B. c., pos.	9/22: Sugar, 0. Albumin, 0. Mod. number of leukocytes.	Vomited once on thirty-seventh day.
28—None	None	9/25: 5,500 W. C.; P., 53; Ly., 47.	9/27: W., pos. 10/1: B. c., neg.	None	Bronchitis (slight).
29—Slight at first; none afterward.	Two	9/30: 5,000 W. C.; P., 70; Ly., 30. 10/17: 6,600 W. C.; P., 65; Ly., 35.	10/3: W., pos. B. c., no report.	None	Bronchitis. Hemorrhage. Vomited.
30—None	None	10/6: 10,000 W. C.; P., 54; Ly., 46.	10/8: W., pos. 10/13: B. c., pos.	None	Headache; poor appetite for first nine days. Then better.
31—Some; tense for two weeks.	Trace on 10/3; none after.	9/20: 10,000 W. C.; P., 74; Ly., 26. 10/6: 10,000 W. C.; P., 60; Ly., 40. 10/8: 15,000 W. C.; P., 45; Ly., 55.	10/2: W., pos. 10/4: B. c., pos.	None	
32—Tense	None	11/12: 14,000 W. C.; P., 60; Ly., 33; Trans., 1; L. mono., 6. 11/26: 6,400 W. C.; P., 50; Ly., 50. 11/28: 6,000 W. C.; P., 48; Ly., 52. 12/6: 12,600 W. C.; P., 61; Ly., 39.	11/15: W., pos. 11/19: B. c., pos.	12/12, 12/27: Sugar, neg. Albumin, neg. Ind., neg.	Acute bronchitis on admission; bronchopneumonia, boils; perirectal abscess.

33—None	None	9/20: 9,800 W. C.; P., 49; Ly., 49; L. mono., 2.	9/22: W., pos. B. c., pos.	9/25:	Albumin, marked trace. Few ly., 0. Gr. casts.	Ring worm on scalp. Red papular eruption. Wassermann positive. Father's Wassermann strongly positive.
34—None on admission; held abdomen tense.	None	9/18: 7,600 W. C.; P., 38; Ly., 53; L. mono., 6; Eo., 3.	9/22: W., pos. B. c., pos.	9/25:	9/23: Albumin, faint trace. Sugar, 0. Ca. oxal crystals.	Delirious, very drowsy and apathetic for six or seven days. Then brighter gradually.
35—Moderate at first. Later soft.	None	9/24: 6,500 W. C.; P., 60; Ly., 40. 9/29: 15,000 W. C.; P., 78; Ly., 22.	9/26: W., pos. B. c., pos.	9/28:	9/25: Albumin, faint trace. No sugar. No casts.	Bronchopneumonia with many small abscesses in right lower lobe.
36—None. Slight tenderness over abdomen on admission.	None	9/19: 7,400 W. C.; P., 51; Ly., 41; L. mono., 5.	9/22: W., pos. B. c., pos.	9/25:	9/13: Albumin, very faint trace. Sugar, 0. Sediment, 0.	None.
37—Complained of abd. pain, but not distended. Cleared up quickly.	None	9/13: 9,000 W. C.; P., 71; Ly., 29. 10/14: 7,000 W. C.; P., 25; Ly., 75.	9/14: W., pos. B. c., pos.	9/16:	9/14: Albumin, faint trace. Few leukocytes. Hyaline casts. Amor. urates. Sugar, 0.	Scoliosis. Otherwise none.
38—None	None	10/13: 6,600 W. C.; P., 66; Ly., 34.	10/13: W., pos. B. c., pos.	10/14:	None	Vomited several time. Uneventful recovery.
39—None	None	9/10: 9,000 W. C.; P., 71; Ly., 29.	9/11: W., pos. B. c., neg.	9/14:	Slight trace of albumin.	None.
40—None during relapse.	None	9/22: 9,500 W. C.; Ly., 59; P., 41. 9/24: 12,500 W. C.; P., 48; Ly., 42.	9/3: W., pos. B. c., pos.	9/6:	9/3: Albumin light cloud. Sugar, 0. 9/14: Albumin, 0. Sugar, 0.	None.
41—Full but not distended.	.....	10/18: 11,000 W. C.; P., 50; Ly., 50. 10/31: 9,000 W. C.; P., 47; Ly., 53. 11/21: 9,000 W. C.; P., 51; Ly., 49.	10/18: W., pos. B. c., pos.	10/20:	11/11, 11/12, 11/14, 11/26: Albumin, sugar and indican, neg.	Boil on right thigh. Vomited once before admission.
42—9/21, slight; 9/24, abd. soft. Soft thereafter.	None	.....	9/5: W., pos. B. c., neg.	9/9:	9/24: Albumin, faint trace. Sugar, 0. Triple phos.	Bronchitis for first three weeks. Nose bleed.
43—Moderate on admission.	10/17: slight. 10/18: 3; 10/19: severe; 10/20: 2; 2 sev.; 1 slight. 10/21: blood in stool; 10/22: hemorrhage.	10/13: 19,000 W. C.; P., 51; Ly., 43; L. mono., 5; Baso., 1. 10/19: 6,600 W. C.; P., 60; Ly., 40. 10/22: R. C., 4,800,000; Hb., 58%. 10/24: 16,000 W. C.; P., 24; Ly., 76. 10/26: 9,500 W. C.; P., 44; Ly., 56. 10/28: 8,500 W. C.; P., 40; Ly., 60; R. C., 3,500,000; Hb., 45%. 11/13: 10,000 W. C.; P., 41; Ly., 51; Hb., 53%; R. C., 2,500,00. 11/17: 6,200 W. C.; P., 36; Ly., 70; Hb., 50%; R. C., 3,000,000. 12/10: Hb., 79%; R. C., 5,700,000; W. C., 17,000; P., 41; Ly., 59.	10/18: W., pos. B. c., pos.	10/20:	11/26: Albumin, trace. Sugar, 0. Indican, ?. 12/8: Albumin, trace. Sugar, 0. Indican, trace. 12/26: Albumin, neg. Sugar, neg. Indican, neg. 10/22: Albumin, mod. trace. Few hyaline and granular casts. Few leukocytes.	Acute bronchopneumonia. Lobar. Acute hepatitis? Cholecystitis? Decubitus. Acute myocarditis.

TABLE 3.—RELATION OF FEEDING TO GENERAL COMFORT AND NERVOUS SYMPTOMS

Case No.	Relapses	Condition of Patient	Medicines	Vaccine
1	None.....	Extremely irritable for three weeks. Appetite never good	Hexamethylenamin.....	None.
2	None.....	Extremely irritable during entire stay, but never really sick. Appetite fair.	None.....	None.
3	None.....	Very nervous and irritable for ten days during which appetite was poor, then better. Condition continued better, as did appetite.	Hexamethylenamin last 7 days.	None.
4	None.....	Restless on admission. Mentality not impaired. Apathetic and irritable for first two and one-half weeks.	None.....	None.
5	None.....	Child was in excellent condition from start to finish.....	None.....	10/30, 200 million.
6	None.....	Apathetic first few days. Later good. Appetite good.....	Hexamethylenamin.....	None.
7	None.....	Very irritable and ill on admission. Appetite fairly good. No nervous symptoms.	None.....	None.
8	None.....	Abdomen held tense on admission. Quite restless for first week. Appetite then good.	None.....	None.
9	.....	Had a few râles at bases.....	.....	None.
10	None.....	Appetite poor for nine days. General condition always good. No nervous symptoms.	None.....	None.
11	None.....	Not stuporous or prostrated on admission. Cried a great deal first few days. Appetite poor ten days then good. No nervous symptoms.	None.....	None.
12	None.....	Appetite poor for five days, then better. No mental disturbance after six days.	Hexamethylenamin.....	None.
13	None.....	Abdomen held tense. Appetite poor for eighteen days. Very quiet for first two weeks. No nervous symptoms.	None.....	None.
14	This attack was a relapse	Very quiet but never had nervous symptoms.....	None.....	None.
15	None.....	Appetite very poor until October 3. Very uncomfortable until September 26, when general condition of nervousness became better. Had headache and was restless.	Caff. Sod. Benz.....	None.
16	None.....	Appetite poor first few days; good for two days then poor again. Picked up finally and had good appetite.	None.....	None.
17	None.....	Appetite poor first seven days; then good. Little sleep first two days; then bright.	Hexamethylenamin.....	None.
18	None.....	Appetite poor but general condition good. Appetite better after four days. No nervous symptoms.	None.....	None.
19	None.....	.....	Pot. Cit. Urot. Calc. Lact.	None.
20	None.....	Always in good condition. Apathetic for a day or two, but then better.	None.....	None.
21	None.....	Had good appetite from start. Never very sick.....	Hexamethylenamin.....	None.
22	None.....	Appetite poor for ten days. Very quiet.....	None.....	None.
23	On fortieth day, but no addition nor symptoms.	September 14, pulse imperceptible. September 16, rational for first time. September 18, irrational again. September 19, brighter.	None.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 150 million; 10/2, 200 million.
24	None.....	Appetite and general condition always good.....	None.....	200 million.
25	None.....	Appetite usually poor; no disturbance of mentality. Well at all times.	Hexamethylenamin.....	None.

26	None.....	Apathetic for first few days. Appetite good always.....	Hexamethylenamin.....	None.
27	Relapse, but general condition remained excellent	Appetite good for first ten days; rather poor on September 15 and 16, but nervous condition at all times good. Up in chair September 20 for two hours till September 26; then to bed again. Appetite good.	Hexamethylenamin.....	None.
28	None.....	Appetite poor for first week. Never very sick.....	None.....	None.
29	None.....	Hemorrhage day after admission and two days later. Transfused and horse serum 30 c.c. injected. Father s blood used in transfusion. Restless and irrational till October 5.	9/30, 15 c.c. horse serum; 10/1, 30 c.c. paternal blood; 10/3, 20 c.c. horse serum; Calc. Lact.; hexamethylenamin.	None.
30	None.....	Refused food for first week. No nervous upsets except headache.	None.....	None.
31	None.....	Delirious for nineteen days with marked meningeal symptoms. Double ankle clonus; stiff neck. Spinal cord tap cells 3. Noguchi negative. Appetite improved October 7.	Hexamethylenamin, salol.	None.
32	None.....	Child was admitted in delirium with meningismus, November 19. Temperature dropped from 105 to 97.6 F. For ten days was nervous and very restless.	Hexamethylenamin.....	None.
33	None.....	Stupid for two days but always had a good appetite.....	Hexamethylenamin last 5 days.	None.
34	None.....	Appetite very poor for seven days then gradually improved. Comfortable first on October 2.	Hexamethylenamin.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 100 million; 10/1, 100 million.
35	None.....	Irrational from second day onward.....	.....	9/28, 50 million; 9/30, 150 million; 10/2, 200 million.
36	None..... ..	Appetite good from start. No nervous disturbance.....	Hexamethylenamin.....	9/11, 50 million; 9/13, 50 million; 9/15, 100 million; 9/17, 100 million; 9/19, 100 million; 9/22, 200 million.
37	None.....	For first two weeks was very apathetic and irrational the first few nights, then better; appetite poor usually; good the last two weeks.	Hexamethylenamin.....	None.
38	None.....	Appetite only fairly good for eleven days, then good. No nervous upsets but cried a great deal on October 14.	None.....	None.
39	None.....	No mental disturbance at any time. Appetite always good	Hexamethylenamin seven days.	None.
40	After three weeks. Made good recovery	Appetite good from start. Only fairly so during relapse for the first three days. Then good again. No nervous symptoms. Somewhat sleepy.	9/17, 9/20, hexamethylenamin.	None.
41	? Had boil.....	Actively delirious on October 24; lasted about one week. Appetite very poor and very weak until October 28; then appetite improved but never very good.	None.....	None.
42	None.....	Appetite rather poor but no nervous disturbances.....	None.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 150 million; 10/2, 200 million.
43	None.....	Child was delirious and weak until early in November. Until November 19, the child did not have one comfortable night; after that slowly improved.	Hexamethylenamin; brandy; Calc. lactate; cod liver oil; wine of iron; Tr. nux vomica.	10/18, 20 c.c.. horse serum; 10/19, 160. c.c. paternal blood; 10/20, 260 c.c. paternal blood 7 a. m., 80 c.c. saline intravenously; 10/22, 1 p. m., 30 c.c. saline intravenously; 10/23, 20 c.c. horse serum; 11/13, 280 c.c. paternal blood intravenously.

partially true, but unless toxic nervous symptoms were present, the fever did not prevent the child from taking a generous diet; as the temperature fell, however, the amount of food taken was greater.

The diagnosis was established by positive Widal reaction in forty-four cases and by positive blood cultures in thirty-six cases. In only one case was there found neither a positive Widal nor a positive blood culture. A complete history of each patient in this series will be found in Tables 1, 2 and 3.

The age range of the patients was as follows: Under 2 years 1 case; from 2 to 6 years, 21 cases; from 7 to 12 years, 26 cases, making a total of 48 cases.

COURSE OF THE DISEASE

The length of time in the hospital averaged twenty-eight days; extremes from thirteen to seventy-nine days. Hand and Gittings report that the average stay in the hospital in their 145 cases was thirty-one days. The ages in their cases ranged from infancy to 12 years, the greater number of the patients being from 4 to 9 years. The day of the disease, as nearly as could be estimated, on which the temperature became normal was found on the average to be the twenty-eighth. Extremes from fifteen to sixty-eight days.

SYMPTOMATOLOGY

The general condition of the patients was remarkably good. The so-called typhoid state occurred in much smaller proportion than under the old methods of treatment, so that the patients did not present, except in rare instances, the typical appearance of typhoid fever.

Abdominal distention.—This was present in only six cases, and in but one of these was it severe or troublesome.

Hemorrhages.—Five patients had, at some time during the course of the disease, blood in the stools. In only two cases were the hemorrhages severe enough to cause any apprehension. In both, blood transfusion was done by the Lindemann method, a procedure which was developed in our wards, and each time with signal benefit. One of the patients, Leonard B., already referred to, was transfused three different times, the amount of blood given being 160 c.c. paternal, the first time, 260 c.c. paternal the second time, and 280 c.c. paternal the third time.

This was the patient who lost the greatest amount of weight and who, during his convalescence, took the largest number of calories we have ever seen recorded, namely, 8,000 in twenty-four hours or 310 calories per kilogram. The reason this enormous amount of food was permitted was because of the boy's constant clamor for a cracker or any form of food to satisfy his gnawing hunger. The eternal call for another cracker induced us to let him eat his fill for a few days, with the result reported. He gained in weight from 45 to 72 pounds in thirty-seven days, an average gain per day of about ¾ of a pound.

Nervous Symptoms.—Thirteen patients were irrational in the early stage of the disease. One who came in during the latter part of the second week of his illness presented the picture of a severe psychosis, a type of nervous disturbance occasionally seen late in the course or following the disease under the old methods of treatment. This nervous state was demonstrated to be a starvation psychosis. After a few days of forced feeding the boy came out of his stupor and delirium and became entirely rational. There is not the slightest doubt that most of the severe nervous symptoms noticed in the late stages of the disease heretofore were due to the starvation and the consquent increase in the toxemia.

Complications.—Pneumonia or bronchopneumonia were present in three cases, and in the only death that occurred the fatal issue proved by necropsy to be due to pneumonia.

Relapses.—Among our cases there were four relapses and one recrudescence, or a little over 10 per cent. Koplik and Heiman report 24 relapses among their 160 cases treated at the Mt. Sinai Hospital, a percentage of 15. Hand and Gittings had 4.8 per cent. of relapses in 145 cases.

Mortality.—There was one death, a mortality of about 2 per cent.

During the course of the fever, 60 per cent. of our patients gained in weight. This was the most remarkable fact about this series of cases; and is, in reality, the reason for this report. Heretofore the loss in weight has been very marked throughout the course of the disease, so that by the time the temperature has become normal, the patient has usually presented the picture of emaciation. The high caloric diet prevents this loss in the great majority of cases or reduces it to a very small amount. If we consider the weight of the patient at the time of leaving the hospital, it is found that 92 per cent. had gained in weight.

In 39 cases there was a gain in weight over that on admission as follows:

Pounds	Cases	Pounds	Cases
¼	1	4	1
½	1	4¾	2
¾	1	5	1
1	1	5¾	1
1½	3	6	1
2	4	6½	1
2¼	3	7¼	3
2½	2	7½	2
3	3	9	1
3¼	3	9¾	1
3½	2	11½	1

CONCLUSIONS

1. It is possible, in the great majority of cases, to feed children suffering from typhoid fever, even during the febrile period, with an amount of food furnishing sufficient calories to maintain or even increase their body-weight.

2. This high caloric diet, besides preventing the emaciation usual in this disease, greatly increases the comfort of the patient, prevents the severe nervous symptoms and lessens the dangers of the disease and of its complications.

DISCUSSION

Dr. Chapin: I would like to ask Dr. Fétra the strength of the cream that he was giving.

Dr. La Fétra: Twenty per cent. cream.

Dr. Churchill: One point struck me about which I would like to ask. Dr. La Fétra spoke of five cases out of forty-eight in which there was blood in the stools, two of those quite severe; and that one of those two patients had a high caloric diet. I don't recall what proportion of typhoid cases in children have blood in the stools; but this seems rather high; in that one severe case, is there any connection between the hemorrhages and the high caloric diet?

Dr. Friedlander: During the period of hemorrhages, was the feeding continued just the same, or was it changed?

Dr. Kerley: I think this is a most valuable contribution, as it will encourage all of us to feed our typhoid patients better. For several years I have fed my typhoid fever patients generously and I am not at all surprised at the results that Dr. Chapin and Dr. La Fétra have published. It has been my practice to give stale bread, gelatin, junket, eggs and cereals, the latter with the addition of sugar and butter. I have never given meat until the third week or later; but all other substances have been my regular dietary for typhoid fever patients. There is one great advantage in getting away from that absolutely whole milk diet; there is much less tympanitis and fewer relapses. The continued diet of milk is one chief means, to my mind, of producing that very distressing symptom tympanitis.

Dr. Morse: Since having typhoid myself, twelve years ago, I have fed my patients on a varied diet. In recent years I have tried to keep the calories up,

but I must say that I have had difficulty in getting the calories as high as Dr. La Fétra and the other speakers did. It doesn't seem to me that it is quite fair to speak of the high caloric feeding of children when they are in the convalescent period. It seems to me we ought to speak of and confine it to the febrile period; because convalescents from typhoid have always had an enormous appetite—they have demanded high caloric feeding, whether the doctor wanted to give it to them or not. We must separate the two periods, the febrile and the convalescent.

Dr. La Fétra: In relation to the diet in the hemorrhage cases, this boy who had the severe hemorrhages, and the others with smaller hemorrhages, were not taking a very high caloric feeding at the time. When the patient is very sick or has marked nervous symptoms, you can not get him to take much food; but just as soon as these symptoms abate there is no trouble. When this boy who had the hemorrhages was able, we urged him to take just as much food as he could; and he took over 107 calories per kilo during the time when his temperature was 103. Dr. Morse is quite right in stating there should be a distinction between the period after the fever is over, and the febrile period itself; for every convalescent typhoid patient will take an enormous quantity of food; and the only reason I mentioned this boy who took the extreme amount in convalescence was to show what sometimes can be done without any ill effect.

THE CALORIC VALUE OF THE DIETS OF SICK INFANTS

J. C. GITTINGS, M.D.
PHILADELPHIA

The problem of the artificial feeding of normal infants, reduced to its simplest terms, is to furnish sufficient nourishment, in digestible form, to supply adequately the infant's nutritional needs. That the amount and form of the nourishment vary with the individual always needs emphasis, but it is undoubtedly true that the average needs of the majority will fall within certain general limits which can be used as a basis for general comparison. The problems of feeding are, of course, greatly modified by illness, but the nutritional needs vary within much narrower limits, and the attempt must always be made to approximate normal requirements as closely as possible, since at no time in life can the body so ill afford starvation as in infancy, especially during the first few months of life.

In determining the normal nutritional requirements of breast-fed babies, a careful comparison of the average intake, with the average growth, and a balancing of both with the composition of the milk, in a large series of cases, will give a fair criterion upon which to judge the amount of the various food elements required. The variable factors are the amount of milk consumed and its composition.

Finkelstein estimates that the average normal breast-fed infant consumes about 150 c.c. per kilo of body weight during the first three months of life; slightly less than this during the second three months, and from 120 to 130 c.c. during the third three months.

Assuming that the percentage of solids (disregarding the salts and ash) in human milk equals approximately 3.5 fat, 6.5 sugar and 1.5 protein, this would give a nutritional requirement of fat, 5.25 grams; sugar, 9.75 grams; protein, 2.25 grams per kilo of body weight during the first six months and fat, 4.38 grams; sugar, 8.13 grams, and protein, 1.87 grams per kilo during the next three months.

Marfan, on the basis of the researches of Camerer, Forster, Uffelman, Veit and Riedel, has calculated the nutritional needs of infants at different ages as follows:

Age	Weight, Kilos	Per Kilo		
		Fat	Sugar	Albumin
3 days	3.0	2.8	2.9	2.4
3 weeks	3.5	5.6	5.7	4.8
7 to 10 weeks	4.0	5.2	5.4	4.5
4 months	6.0	4.5	4.6	3.8
18 months	9.0	4.0	8.9	4.4
28 months	10.0	2.7	15.0	3.6
To which may be added by computation—				
8 months	6.85	4.36	5.83	3.97
12 months	7.70	4.21	7.06	4.14

The difference between the two series is found chiefly in the relative amounts of sugar and protein, their sum total being about equal.

In determining the nutritional requirements of bottle-fed babies, three methods have been employed, viz.: (1) Metabolism experiments, (2) determination of the energy exchange with the calorimeter, (3) "statistical method," in which the intake is balanced over long periods of time with the output, calculated from occasional estimations of the excreta.

The difficulties inherent to metabolic investigations upon infants, and the great variation in the energy output, depending upon the activity or non-activity of various bodily functions and exercise, limit the value of both of these methods, at least so far as the present-day technique has been developed. Howland believes that by clinical observation and not by physiological or chemical determinations will the superiority or inferiority of a diet or method of feeding eventually be established, with the necessary proviso that the clinical observation must be prolonged, thorough and judicious. He also points out that the statistical method can be applied much more accurately to bottle-feeding than to breast-feeding, although the opportunity seems to have been largely neglected.

Until a more certain basis is established we may accept the figures given for the food requirements of breast-fed infants as a fair minimum for the artificially-fed child, with certain modifications to suit individual needs.

Disregarding the salts, about which discussion is rife, the protein undoubtedly is the most important food constituent during infancy. Its three chief functions are: (1) To furnish building material for the

tissues of the body, (2) to repair daily waste of tissue, and (3) to furnish heat by combustion.

The first function is of prime importance, especially during the first few months of life. Even if insufficient amounts of protein are fed, and weight remain stationary, or actually diminish, growth in length will take place, although retarded. The repair of daily waste increases with age and activity, at the same time that storage, for purposes of growth, is diminishing. Meara estimates that in the first two weeks of life 75 per cent. of the protein is stored; at two months, 40 per cent., and at five months, 23 per cent.

The third function, heat production, is incidental, since protein is a wasteful source of supply for heat; approximately 30 per cent. of its caloric value is wasted in the physiological processes involved in the digestion and assimilation of protein (Meara).

Of the metabolic changes which fats and sugars undergo in the economy, less is known than in the case of protein, but their two important functions are the production of energy and protection of the body protein. No diet poor in both fat and carbohydrates will produce satisfactory gain in weight, while too great deprivation results in emaciation from the consumption of the body tissue, since protein alone, even in large amounts, is insufficient to supply both energy and food for tissue.

The carbohydrates are more important than the fats as protein savers, and assume increased value in the case of bottle-fed infants, in whom the digestion of fat frequently offers such difficulties.

On the other hand, judging from clinical observation, it is undoubtedly true that fat plays an important rôle in increasing weight, since those babies gain best who can successfully digest liberal amounts of fat. Contrary to the old teaching, there is increasing evidence that infants can digest cow's milk protein as well as that of human milk, while nitrogen retention on a cow's milk diet is equally good, or even better, than in human milk. While an excess of protein probably entails some dangers, as seen in the interesting experiments of Holt, Lavene and their assistants, where high temperature was regularly produced by feeding a high "synthetic" protein mixture, the danger is less than with an excess of fat or sugar.

Accepting the figures given for the nutritional requirements based on Finkelstein's estimated intake, the caloric equivalent of a breast-milk diet may be worked out as follows, using Rubner's factors of 9.3

calories per gram of fat and 4.1 calories per gram, each, for sugar and protein:

First three months—

Fat	5.25 grams × 9.3	= 48.83
Sugar, Protein	12 grams × 4.1	= 49.20
		98.03 calories per kilo.

From the third to sixth month the proportion is slightly less.

Sixth to ninth month—

Fat	4.38 grams × 9.3	= 40.73
Sugar, Protein	10 grams × 4.1	= 41.00
		81.73 calories per kilo.

In other words, the caloric requirements of breast-fed babies vary from 100 calories per kilo, during the first month, to 80 calories per kilo at the end of lactation (nine to twelve months). Upon these figures most authorities approximately agree. On the ground that the assimilation of cow's milk requires more work by the infant's organism than is the case when breast-milk is fed, Rubner places the energy quotient (calories per kilo) at 120 during the first few months. Unless the fat percentage in a mixture be quite high, it is impossible to even approximate such a figure as 120, and, with a view to the risk of inducing fat indigestion during the first months of artificial feeding, many authorities accept a lower energy quotient, from 100 to 110 calories per kilo, during the first three months of life.

For practical purposes the factor of body weight is used in determining caloric requirements. A better index, however, is body surface, since loss of heat is proportionate to surface area, while the relation of area to weight is proportionately greater in small than in large bodies. In infants of equal surface area, but different weights, heat radiation is greater in the one weighing least.

In comparing infants' and adults' diets, it is found that infants require a much more liberal supply of all the food constituents in proportion to their weight and a much more liberal supply of fat and water compared to their need for protein.

For example,

	Fat	Sugar	Protein	
Infant, 3 months, 5 kilos.....	5.25	9.75	2.25	per kilo body weight
Adult, 150 kilos	.71	5.7 to 7.1	1.43	per kilo body weight
(Meara)				

As Waller points out, the infant requires a greater amount of protein than the adult, in proportion to body weight, but in proportion to body surface the amount is approximately the same.

In stating caloric requirements, allowance must be made for the error due to great deficiency in weight, and the figures for atrophic infants must be increased above the average. Tabulating these requirements, we have,

		Per Kilo
First 3 months	45 to 50 calories per pound body weight	(100 to 110)
3 to 6 months	40 to 45 calories per pound body weight	(85 to 100)
6 to 12 months	35 to 42 calories per pound body weight	(75 to 90)
12 to 24 months	30 to 40 calories per pound body weight	(65 to 85)

The value of estimating the caloric values lies in the fact that in no other way do we receive such striking proof of deficiencies or excesses in the dietaries. While a fairly wide latitude may be allowed in applying general values in any particular case, and while we should never allow theoretical considerations to completely offset careful clinical deductions, the fact remains that, in the majority of cases, a marked caloric deficiency or excess continued over a length of time is reasonably sure to be followed by nutritional or digestive disturbances. Finkelstein finds that an energy quotient of 70 calories during the first half year results in stationary weight.

The maximum figures of safety are more difficult to determine, as infants show a great variation in their ability to handle excessive amounts of food. Since fat furnishes the highest caloric values, and a normal caloric average is difficult to maintain without a fair percentage of fat in the mixtures, it is reasonable to assume that the maximum figures given in the last table cannot be safely exceeded for any length of time without danger of causing fat indigestion. On the other hand, too close adherence to the principles of caloric feeding entails certain risks, especially in feeding sick infants. The primary object of dietetics in digestive disturbances must be to fit the food to the digestive functions and caloric values must be used merely as a guide to nutritive requirements, not as compulsory dietetic indications. Again, in the attempt to substitute one food constituent for another, which is disagreeing, in order to build up caloric values, due regard must be held for the safe limits of such increase. Probably protein can be increased far beyond the usual average with greater safety than either fat or sugar, but every case must be decided upon its merits and general rules are hard to formulate.

Again, the tendency at times may be to lay too much stress upon clinical data. For example, in the attempt to adjust the diet to the point at which the stools present a normal appearance, the diet may be kept far below the limits of safety. The caloric value of the diet furnishes at once the measure by which we can estimate the risk of starvation and weigh it in the balance against the risk of still further increasing indigestion.

The following statistical investigation of the dietaries of sick infants was made with a view to obtaining a general impression of my own methods, judged by the energy quotient. No deductions can be drawn from it as to the needs of well infants. Certain well-known clinical facts were corroborated and, in the main, the truth of the danger to the infant's organism of under-feeding was clearly substantiated. The length of stay in the hospital was too short to prove any of the dangers of over-feeding, judged on a caloric basis. The study certainly offers a stimulus to greater care in the selection of dietaries, since the percentage of deficiency in some instances was startlingly large and suggests the possibility that a more judicious substitution of food ingredients might have prevented some of the loss. The cases were those seen in my service for the last three years at the Children's Hospital. None was included which was not under treatment for at least one week, the average being three weeks. This eliminated many fatal cases, dying within one week. The majority of patients were suffering from gastrointestinal or nutritional disorders. A small group of miscellaneous diseases was included for comparison. The caloric value of the diets was calculated on the basis of food consumed, careful record of which is routinely noted, and on the caloric value of foods as determined by Atwater and Bryant's tables. No attempt was made to select cases, and only those were excluded which did not answer to specifications of age, diagnosis or length of stay. A few cases were not included because of very frequent changes in diet which necessitated an expenditure of time in calculation that the value of the work did not warrant.

In each case the total caloric value of the food consumed was calculated and the average per day used as a factor in determining the percentage of deficiency (or excess) of that diet. Two standards were used for comparison. First, the caloric requirements of each child, calculated on the basis of his weight on admission; Second, the caloric

requirements for an infant of average size and of the same age. The former did not furnish a true index of the caloric needs of each case, as the initial weight often rapidly increased, which entails a corresponding increase in the daily caloric allowance. This initial weight standard furnished an absolute minimum for each child, however, while the normal weight standard furnished a caloric optimum. In determining this normal weight standard, the following weight chart was used. While the figures are somewhat arbitrary, they cannot be considered excessive and comprise the results of a former careful compilation by Dr. Judson and the author. In computing the caloric requirements for the various weights in the table, the factors used were the highest figures in the table, viz: First three months, 50 calories; three to six months, 45 calories; six to twelve months, 40 calories; twelve to twenty-four months, 40 calories, as each child was below the normal weight for its age.

In computing the first standard, namely, the caloric requirements based on the weight on admission, the same values were used.

	Weight of Average Bottle Fed Infant	Total Calories Required
At end of 1 month	8 pounds	400
At end of 2 months.....	9½ pounds	475
At end of 3 months.....	11 pounds	550
At end of 4 months.....	12½ pounds	575
At end of 5 months.....	13½ pounds	600
At end of 6 months.....	14½ pounds	615
At end of 7 months.....	15½ pounds	630
At end of 8 months.....	16 pounds	645
At end of 9 months.....	16½ pounds	660
At end of 10 months.....	17 pounds	680
At end of 11 months.....	18 pounds	720
At end of 12 months.....	19 pounds	760
At end of 18 months.....	21 pounds	840
At end of 24 months.....	24 pounds	960

The total number of cases tabulated was 125. Of these there were 35 cases of infantile atrophy, 72 cases of acute or chronic gastro-intestinal disease and 18 of miscellaneous diseases, including pneumonia (8), bronchitis (1), ulcerative stomatitis (2), typhoid fever (1), scurvy (1), syphilis (2), tuberculous meningitis (2), and pylorospasm (1).

In explanation of the nomenclature, the cases of "atrophy" gave a history of prolonged nutritional disturbance, with an average weight of 7 pounds, 14 ounces for an average age of 4.6 months on admission. Cases included under the head of gastro-intestinal disease gave evi-

TABULATED CASES

Classification	Number of Cases	Average Age in Months	Average Days in Hospital	Average Weight on Admission	Normal Weight for Same Age	Average Loss or Gain in Ouncees	Mortality	Percentage of Deficiency in Caloric Value of Diet Taken	
								Compared with that Indicated by Weight on Admission	Compared with that for Normal Child of Same Age
				Lbs. Oz.	Lbs. Oz.	Ounces	Per Cent.	Per Cent.	Per Cent.
All Cases.....	125	7.3	26	10—11	15—11	+ 5.4	20	10.9	36.4
Deaths.....	26	5.0	20	8—11	13— 8	— 14	...	31.2	57.4
Recoveries.....	99	8.0	27½	11— 6	16	+ 10½	...	7.2	30.9
Atrophy Cases *....	10	4.5	33	8— 2	13	+ 18½	10	17.4 ‡	28.6
Uncomplicated Atrophy Cases †....	25	4.6	28	7—12	13— 2	— 2⅓	52	6.4	45.1
Complicated Gastro-Intestinal *..	32	7.5	25	11— 4	15—12	+ 13	0	11.9	34.2
Disease, uncomplicated Gastro-Intestinal †..	40	7.8	25½	11— 1	15—14	— ½	15	23.7	44.3
Disease, complicated Miscellaneous.....	6	9.8	21⅓	12	16—14	+ 13⅓	16.7	14 ‡	3.0
Tubercular Meningitis.....	2	11.5	22½	13—14	18— 7	+ 6½	100	10.8	33.3
Pneumonia.....	8	12.8	22	15—12	19— 6	+ 2⅕	25	15.1	30.7
Typhoid Fever.....	1	16.0	45	16— 8	21	+ 52	0	78.2 ‡	45.2 ‡
Pylorospasm.....	1	1.0	21	6— 6	8	— 1	0	10.6	28.5

* No fever, diarrhea or vomiting. † Fever, diarrhea or vomiting, one or more. ‡ Excess.

Occasional rectal temperature to 100 F. was disregarded.

Continuous rectal temperature to 100 F. or over 100 for more than 10 per cent. of the days was accounted "fever."

Stools numbering under four per day were disregarded. Over four, were accounted diarrhea.

Regurgitation or vomiting totaling less than 33 per cent. of the number of days in the hospital was disregarded; their occurrence on over 33 per cent. of the days was accounted vomiting.

dence of more or less severe involvement of the gastro-intestinal mucosa or had been sick only a short time.

Of the 125 cases, the average weight on admission was 10 pounds, 11 ounces for an average age of 7.3 months. The average nutritional deficiency, therefore, was high.

The noteworthy features of the tabulated results are as follows:

The percentage of deficiency in caloric requirements based on weight on admission (minimum value) was reasonably small, but the small gain (⅕ of an ounce a day for "all cases," ⅖ of an ounce a day for "recoveries") suggests that it might have been possible to exceed the caloric minimum with benefit.

The complicated atrophy cases showed about the same deficency as the "recoveries," but in the uncomplicated atrophies and in the miscellaneous group, where, as a rule, no contraindications to full feeding existed, the diet exceeded the minimum requirements and the gain was proportionately large.

In the gastro-intestinal group a similar difference was noted, but the uncomplicated cases showed an actual deficiency instead of excess and received less food than careful attention to caloric requirements would have indicated.

The cases of tuberculous meningitis showed an average *gain* in weight because of forced feeding by gavage in one of the cases, which had received practically no nourishment for three weeks prior to admission. Forced feeding also reduced the percentage of caloric deficiency.

The figures for the cases of pneumonia showed the retarding effect of fever and toxemia *per se* upon the ability to take nourishment, while these effects in the case of typhoid fever were offset by the longer stay in the hospital during convalescence. The tremendous excess of nourishment in the case of typhoid fever also accounts for the excellent gain in weight (½ pound a week) and emphasizes the value of judicious forced feeding in typhoid fever during childhood.

The deficiency in caloric values of all of the diets compared with the requirements for normal children of the same age forms an interesting comparison. It must be judged with a view to the deficiency in weight on admission compared with that of the normal child. For example:

	Percentage of Admission Weight Deficiency Compared with Same Age	Percentage of Caloric Deficiency Normal Child,
1. All cases	32 per cent.	36 per cent.
2. Deaths	36 per cent.	57 per cent.
3. Recoveries	29 per cent.	31 per cent.
4. Atrophy cases (complicated)	41 per cent.	45 per cent.
5. Gastrointestinal (uncomplicated)	29 per cent.	34 per cent.
6. Gastrointestinal (complicated)	30 per cent.	44 per cent.
7. Atrophy cases (uncomplicated)	37 per cent.	29 per cent.
8. Miscellaneous	29 per cent.	3 per cent.

From 1 to 6, inclusive, show a greater deficiency in calories than in weight, while the uncomplicated atrophies and the miscellaneous group show the reverse. Again the uncomplicated gastrointestinal group shows a caloric deficiency which probably was not justified.

The difficulty in feeding sufficiently high caloric values to very young infants is suggested in the following table:

			——Caloric Deficiency——	
Age	Number of Cases	Average Gain or Loss in Ounces Ounces	Based on Weight on Admission Per Cent.	Based on Normal Weight for the Same Age Per Cent.
1 to 3 months	34	0	26	48
3 to 6 months	35	+5½	7	39
6 to 12 months	34	+7	7	30

Finally, the general relationship of caloric requirements to loss or gain in weight may be seen in the following summary:

			——Caloric Deficiency——	
Age	Number of Cases	Average Gain or Loss in Ounces Ounces	Based on Weight on Admission Per Cent.	Based on Normal Weight for the Same Age Per Cent.
Cases which lost weight	44	−15⅓	32	52.8
Cases which gained weight	78	+17⅓	1.3 excess	27.9
Cases with stationary weight	3	0	28.1	41.6

In closing I wish to express my indebtedness to Dr. Henry S. Kinloch, intern at the hospital, for tabulating the diets, and to Dr. A. Graeme Mitchell for calculating caloric values of diets, etc.

CONCLUSIONS

The value of a conclusion based on general averages cannot always be applied to the individual case, but if the caloric value of the individual diet persistently falls much above or below the average, harm will result in the majority of cases.

Caloric estimations, therefore, should be used merely as danger signals, but not necessarily as sign-posts. Due regard must be paid to the component parts of the diet which go to make up caloric values, and especially to the clinical effects of diet upon digestion and health.

Theory should never outweigh practice, in the best interest of the child, but in the long run sound theory usually will be found to agree with practice.

3942 Chestnut Street.

STUDIES IN THE RESPIRATORY EXCHANGE OF INFANTS *

FRANCIS G. BENEDICT AND FRITZ B. TALBOT
BOSTON

An extensive study of the gaseous exchange of infants has been in progress for three years in connection with the Nutrition Laboratory and with the Massachusetts General Hospital. Pending the development of a method for determining the oxygen consumption directly — a factor that we deemed of very great importance — a preliminary communication[1] was made in 1912. Since the appearance of this article, the apparatus and technic have been perfected so as to include direct determinations of the oxygen consumption. An apparatus has been constructed, tested and installed in a special room in the Massachusetts General Hospital, and except during the summer months, studies of the gaseous metabolism of infants have been made daily since January, 1913. Although the investigation is still in progress, sufficient data have been accumulated to justify the presentation of material *in extenso* in a publication of the Carnegie Institution of Washington.[2] A full description of the apparatus and technic, the clinical history of the infants studied and the statistical data for each observation are given in that publication for permanent record. It is our purpose in this communication to present first, a complete collection of the literature bearing on this subject up to the present date, and second, an amplification of our data by a new series of observations and finally an abstract of our results.

PREVIOUS OBSERVATIONS ON THE GASEOUS METABOLISM OF INFANTS

The earliest record that we have been able to find of the measurement of the gaseous metabolism of an infant is that reported by

* From the Nutrition Laboratory of the Carnegie Institution of Washington and the Children's Department of the Massachusetts General Hospital, Boston, Mass.

1. Benedict and Talbot: Some Fundamental Principles in Studying Infant Metabolism, Am. Jour. Dis. Child., 1912, iv, 129.

2. Benedict and Talbot: The Gaseous Metabolism of Infants with Special Reference to Its Relation to Pulse-Rate and Muscular Activity, Carnegie Institution of Washington, Publication 201, 1914.

J. Forster of Munich, in 1877† In an effort to explain the well-known fact that children consume a larger amount of food in proportion to their body-weight than do adults, this investigator made determinations of the carbon dioxid excretion in fourteen experiments on children varying in age from 14 days to 9 years. His results all showed that 10 or 12 gm. of carbon dioxid were given off per hour for every 10 kg. of body-weight. With adults on the same basis, the carbon dioxid given off under conditions of rest and approximate hunger was 4 to 5 gm. per hour; with an average diet, 5 to 6 gm.; and with food and work, 7 gm. The author points out that the youthful organism, even in the condition of hunger, produces per unit of weight at least twice the amount of carbon dioxid which is produced by the adult body. The fact is also recognized that the infant can develop a considerable amount of work.

The experiments were made with the large Pettenkofer-Voit respiration chamber in Munich, but the protocols were never published, and aside from the statement that the children were at rest, no further details are given as to the muscular activity or the pulse-rate.

In 1885 Richet,[3] in describing his calorimeter, states that he has two chambers, one of which, having a capacity of about 1,500 liters of air, is used for experiments with infants. He cites an experiment with an infant of 9 kg., who was in the chamber for one hour and ten minutes, and gives protocols for another experiment, one hour in length, presumably with the same infant. In summing up his averages he refers to numerous experiments on infants weighing from 6 to 9 kg., and includes observations made with environmental temperatures ranging from 18 C. to 25 C. He concludes that the infant produces on the average 4 calories per kilogram of body-weight per hour. Richet discusses especially the relationship between the body-surface and the heat-production.

Two years later, Langlois[4] conducted experiments on children with Richet's calorimeter, in which only the heat-production was measured. From 17 controlled experiments, all with infants weighing about 7 kg., Langlois concludes that the heat-production is increased as the environmental temperature is lowered. As a result of a study of the influence

† Forster: Amtl. Ber. d. 50. Versamml. deutsch. Naturf. u. Aerzte in München, Munich, 1877, p. 355.

3. Richet: Archiv. de physiol. norm. et. path., 1885, Series 3, xv, 237.

4. Langlois: Centralbl. f. Physiol., 1887, i, 237.

of the time of day on the heat-production, he also concludes that there are two maximum values at approximately 11 a. m. and 3 p. m., corresponding to the values for the absorption of oxygen found by Fredericq.[5]

Langlois' discussion of the relationships between the heat-production and the body-weight and the heat-production and the body-surface is of special interest in connection with our research. As will be seen from his figures given in Table 1, the smaller the child the larger was the heat-production per kilogram of body-weight. The author points out, however, that if the heat-production and body-surface are compared, as is done in Table 2, the uniformity is remarkable. He gives a very unsatisfactory explanation of his unit of surface, but brings out the fundamental idea that the heat per unit of body-surface is essen-

TABLE 1.—RELATIONSHIP BETWEEN HEAT-PRODUCTION AND BODY-WEIGHT OF INFANTS (LANGLOIS)

Body-Weight (kg.)	Heat-Production per kg. of Body-Weight (cals.)
Two children 1.8	6.40
Child 2.5	4.80
Children from .....3.0 to 4.0	4.20
Children from .....7.0 to 8.0	4.12
Children from .....9.0 to 10.0	3.93

tially the same for an infant as for an adult weighing 65 kg., namely, 14 to 17 calories. No information is given regarding the muscular activity, the age, or the pulse-rate of these infants.

In another paper, Langlois[6] refers to Richet's observations on normal children weighing from 7 to 10 kg. with a heat-production of approximately 4,000 calories per kilogram per hour, and reports his own results with sick infants in which he finds a direct relationship between body-temperature and heat-production. Infants having temperatures below 37.5 C., which he takes as normal, had a heat-production less than 4 calories per kilogram of body-weight per hour, while those with temperatures above 37.5 C. had a higher heat-production; thus, with a body-temperature of 35.5 C., the heat-production was equal

5. Fredericq: Arch. de Biol., 1882, iii, 731.

6. Langlois: Compt. rend de l'Acad. des Sci. de l'Institut de France, 1887, civ, 860.

to 2,900 calories, while with a body-temperature of 40.5 C., it was equal to 4,600 calories.

Langlois' calorimeter was subsequently used by Variot and Saint-Albin[7] in studying the calorimetry of atrophic infants. The tests of this calorimeter published by Saint-Albin[8] show a possible error of plus or minus 10 per cent., thus indicating that the apparatus can hardly be considered an instrument of precision. As Saint-Albin points out himself, his check tests differ considerably from those of Langlois.

Variot and Saint-Albin studied a large number of atrophic infants. Their conclusions, reported by Saint-Albin, are especially interesting

TABLE 2.—RELATIONSHIP BETWEEN HEAT PRODUCTION AND BODY-SURFACE OF INFANTS (LANGLOIS)

Body-Weight kg.	Body-Surface	Heat-Production		
		Per kg. of Body-Weight cals.	Per Unit of Surface cals.	Per Sq. Meter of Body-Surface † cals.
10	9.142 *	3.920	17	1,690
9	2.106	3.900	16	1,620
7	1.778	4.120	16	1,580
6	1.638	4.200	15	1,500
4	1.135	4.300	15	1,370
2	0.780	6.000	15	1,510

* This figure is quoted from Langlois and as his discussion of body-surface is very confusing, it is impossible to make a correction, which is obviously much needed.

† As calculated by Camerer using Meeh's formula (Camerer: Der Stoffwechsels des Kindes, Tübingen, 1896, p. 109).

in connection with this report of our researches, since they show that (using their terminology) out of 33 atrophic infants, there were 11 *"hyperrayonnants,"* 16 *"hyporayonnants"* and 6 *"rayonnants normalement."*[9]

7. Variot and Saint-Albin: Bull. Soc. de pédiat., 1903, v, 246 and 307. See, also an extensive discussion of these researches in the thesis by Saint-Albin: Etude sur la calorimétrie des infants atrophiques, Paris, 1904, No. 295.

8. Saint-Albin: Etude sur la calorimétrie (Note 7), p. 25.

9. Saint-Albin: Etude sur la calorimétrie (Note 7), p. 39.

Of the numerous forms of calorimeters reported to the French scientific societies by d'Arsonval, one[10] was employed by Bonniot[11] in 1898 for a study of the heat-production of infants with temperature disturbances, but he found no regular relationship between heat radiation and rectal temperature. A detailed presentation of Bonniot's results may be found in his thesis.[12]

The most recent contribution from French laboratories on direct calorimetry with which we are familiar is that of Variot and Lavialle[13] in 1912. In this interesting communication, in which the fundamental principles of infant calorimetry are well considered, the authors state that they used the modified form of the d'Arsonval calorimeter which was calibrated by electrical resistance. The gaseous metabolism was not studied and no statement was made as to the muscular activity of the infants. The authors conclude that the heat-output of infants increases in proportion as the weight decreases and lay great emphasis on the effect of clothing on radiation. They likewise believe that the supply of adipose tissue may materially modify the radiation.

Mensi[14] of Turin, without stating the apparatus employed or even the fundamental principle, reports a series of observations on five newborn infants varying in age from 6 hours and 5 minutes to 7 days, 17 hours and 54 minutes. In these experiments the oxygen consumption was determined as well as the carbon dioxid production. The results are given in Table 3. The statement is made that the infants were quiet in each case, but no pulse records are given.

A very interesting series of experiments on infants was carried out by Scherer in the institute of Professor Marês in Prague[15] with an apparatus on the Regnault-Reiset principle, the oxygen being supplied from a bomb. The author states that the infants found themselves in

10. See note on this particular calorimeter by d'Arsonval: Mem. Soc. de biol., 1898, p. 248.

11. Bonniot: Mém. Soc. de biol., 1898, p. 249. For a critique of the Richet and d'Arsonval calorimeters, see Bonniot: Calorimétrie infantile état de la question, Clinique Infantile, 1906, iv, 289.

12. Bonniot: De l'hyperthermie dans la fièvre; essai de calorimétrie clinique, Paris, 1900, No. 419.

13. Variot and Lavialle: Bull. and mém. Soc. méd. d. hôp. de Paris, 1912, Series 3, xxxiii, 410. See, also, Clinique Infantile, 1912, x, 229, and Report of the Congrés National des Gouttes de Lait tenu à Fécamp les 26, 27, et 28 Mai, 1912, p. 79.

14. Mensi: Gior. d. r. Accad. di med. di Torino, 1894, lvii, 301.

15. Scherer: Jahrb. f. Kinderh., 1896, new series, xliii, 471.

TABLE 3.—Summary of Experiments on Respiratory Exchange of New-Born Infants (Mensi)

Age d., h., m.	Sex	Body-Weight kg.	Length of Experiment minutes	Oxygen Consumed			Carbon Dioxid Produced				Respiratory Quotient
				Total c.c.	Per Minute c.c.	Per Kg. per Min. c.c.	Total c.c.	Per Min. c.c.	Per Kg. per Minute		
									c.c.	mg.	
0 6 5	M.	2.70	173	5,707	32.9	12.18	4,174	24.1	8.92	16.08	0.73
1 2 47	M.	3.00	173	6,470	37.4	12.46	3,844	22.2	7.40	13.34	0.593
3 1 55	M.	2.92	159	6,216	39.09	13.38	3,442	21.64	7.41	13.36	0.55
3 7 22	F.	2.47	171	5,463	31.9	12.91	3,586	20.9	8.46	15.25	0.655
7 17 54	M.	2.32	149	4,997	33.5	14.43	2,979	19.9	8.57	15.45	0.59

"complete physiological condition" inside the chamber. In this series, fifty-five experiments were made in the spring and summer and thirty experiments in the winter, each one being about two hours long. No information is given regarding the activity of the infants or the pulse-rate. The fact that the average respiratory quotients were considerably below 0.6 points strongly to an error in the method. The author concludes that the intensity of the respiratory exchange is dependent on the body-weight and is inversely proportional to it.

Two years later the classic monograph of Rubner and Heubner[16] appeared. In discussing the earlier observations of Forster, they add the significant fact that Forster's experimental periods were but one hour long. As their own work was done with the Pettenkofer chamber, they criticize adversely the closed circuit apparatus used by Scherer, and particularly the fundamental principle of using experiments with short periods, their paper setting forth fully the arguments in favor of the long experimental period and the Pettenkofer type of respiration chamber as compared with the short period and the Regnault-Reiset chamber. The Pettenkofer chamber, which had previously been described,[17] was slightly modified for the studies of Rubner and Heubner, a small chamber being used.

The fundamental question studied by Rubner and Heubner was the nourishment of an infant from a practical point of view; and they were accordingly more interested in the average daily requirement of an infant for nourishment than in the physiologic fact of the minimum requirement for comparison with other individuals. The subject—a "normal" infant—was 9 weeks old at the time of the observation and weighed 5,220 gm. The infant was placed in the respiration chamber and removed and fed from six to eight times each day, the time thus lost being carefully recorded. Ocular observations of the muscular activity were made and a general impression for each day recorded. For much of the time, the infant was awake but not crying. On the basis of twenty-four hours the authors found a difference of 22 per cent. between the minimum and maximum carbon dioxid production. They state that this difference is due to the unequal activity of the infant, emphasizing especially the fact that disturbance of sleep during the night influences the total daily average of the metabolism.

16. Rubner and Heubner: Ztschr. f. Biol., 1898, xxxvi, 1.
17. Wolpert: Arch. f. Hyg., 1896, xxvi, 32.

Using the formula of Meeh[18] and a body-weight of 5.1 kg., they compute the body-surface to be equal to 3,500 square centimeters and find a carbon dioxid production of 13.5 gm. per square meter of body-surface per hour. Comparing this value with those values found with adults, they state that the infant excreted less carbon dioxid per square meter of body-surface than did the adults, and explain this by the fact that the infant was asleep a part of the time while the determinations with adults were made only when the subjects were awake. Having pointed out that their results contradict those of Sondén and Tigerstedt,[19] which showed an increased production of carbon dioxid in youth, they emphasize the fact that the carbon dioxid is essentially proportional to the body-surface with human individuals of any size.

TABLE 4.—RESULTS OF EXPERIMENTS WITH A NORMAL INFANT AND AN ATROPHIC INFANT. (RUBNER AND HEUBNER)

Description of Subject	Food	Period	Calories per Square Meter of Body-Surface
Normal	Breast-fed	...	1,006
	Cow's milk.....	I	1,143
	Cow's milk.....	II	1,233
	Cow's milk.....	III	1,378
Atrophic	Cow's milk.....	I	1,090
	Cow's milk.....	II	1,171
	Meal	...	1,036

Shortly after the publication of their investigations with a normal, breast-fed infant, Rubner and Heubner[20] reported a comparative study with a normal and an atrophic infant, neither being breast-fed. This study was carried out on the same plan as that used for the former experiment. The "normal" infant weighed 7.57 kg., was 7½ months old and appeared to be in good health. She was fed on milk and milk-sugar and throughout the observation was said to be in general *recht ruhig*. The results were compared with those obtained with the breast-fed infant in the previous experiment, the hourly excretion of carbon dioxid per square meter of body-surface being but 13.5 gm. for the

18. Meeh uses the formula $S = 11.9 \sqrt[3]{w^2}$. (Ztschr. f. Biol., 1879, xv, 425.)
19. Sondén and Tigerstedt: Skandin. Arch. f. Physiol., 1895, vi, 1.
20. Rubner and Heubner: Ztschr. f. Biol., 1899, xxxviii, 315.

TABLE 5.—Results of Experiments on the Respiratory Exchange of Atrophic Infants (Poppi)

Name	Date 1899	Duration hrs.,min.	Body-Weight kg.	Age mos.	Carbon Dioxid Produced			Oxygen Consumed			Respiratory Quotient
					Total c.c.	Per 24 Hours liters	Per Kg. per Min. c.c.	Total c.c.	Per 24 Hours liters	Per Kg. per Min. c.c.	
P. L.....	July 11	1 15	3.425	9	2,166	40.62	8.237	2,084	40.02	8.114	1.015
N. B.....	July 20	2 00	3.865	10	3,900	46.80	8.409	3,820	45.84	8.236	1.021
C. F.....	Nov. 10	2 00	5.500	16	4,125	49.50	6.25	4,621	55.452	7.002	0.893
A. F.....	Nov. 12	2 00	3.465	7	3,177	38.13	7.642	3,027	36.324	7.280	1.050
M. G.....	Nov. 15	2 00	5.450	12	4,071	48.852	6.225	4,186	50.232	6.40	0.973
F. G.....	Dec. 8 1900	2 00	2.780	3½	2,667	32.004	8.07	2,642	31.704	7.92	1.019
E. N.....	Feb. 3	2 00	3.940	4	3,009	36.108	6.364	3,223	38.676	6.818	0.933

breast-fed infant, which weighed but 5 kg., and 17.3 gm. for the artificially fed infant, which weighed 7.6 kg.

The second portion of the paper deals with the metabolism of the atrophic infant, artificially fed with cow's milk and *"Kindermehl."* Their results are given in Table 4. The authors conclude that there was nothing abnormal in the metabolism of the atrophic infant.

TABLE 6.—METABOLISM OF INFANTS COMPARED (RUBNER AND HEUBNER)

Subjects and Diet	Body-Weight kg.	Calories per Square Meter of Body-Surface per Day
Atrophic child (cow's milk).....	3	1,090
Breast child	5	1,006
Child (cow's milk)	8	1,143
Child (breast, of this experiment)....	10	1,219

The first extensive study exclusively with atrophic infants was made by Poppi.[21] A respiration apparatus of the closed-circuit type was probably used, as both the carbon dioxid production and the oxygen consumption were measured, though little is said of the

TABLE 7.—METABOLISM OF AN INFANT (SCHLOSSMANN AND MURSCHHAUSER) (PER SQUARE METER OF BODY-SURFACE PER HOUR)

	Oxygen Consumption gm.	Carbon-Dioxid Production gm.
Average during eight hours' sleep.....	11.0	13.78
Shortly after feeding.....	11.88	15.52
Three hours after feeding.....	10.42	12.68
Waking and sleeping.....	12.85	15.75

method. An abstract of the results obtained with seven infants is given in Table 5. The respiratory quotients all seem unusually high, and this fact throws doubt on the accuracy of the research. It is probable, however, that the carbon dioxid determinations are well within the

21. Poppi: Il ricambio materiale e il ricambio respiratorio nell'atrofia infantile, Bologna, 1900.

limits of accuracy, as is usual with methods of this type. From the protocols of one of Poppi's studies, it appears that the experiments were each two hours long, but no estimations are given regarding the muscular activity or the pulse-rate.

In 1904 Rubner and Heubner[22] reported another series of experiments covering a period of five days. The subject was a breast-fed infant, 5½ months old and weighing 9.7 kg. Notwithstanding the apparently large changes in the activity from day to day, the investigators found that the carbon dioxid output on the last four days was fairly constant—a fact which puzzled the authors, who suggest a compensatory influence in the life of the infant. They compare the

TABLE 8.—RESULTS OF FASTING EXPERIMENTS WITH INFANTS DURING APPROXIMATELY ABSOLUTE REST. (SCHLOSSMANN AND MURSCHHAUSER)

Subject	Age Days	Weight kg.	Body-Surface Square Meter	Carbon Dioxid per Square Meter per Hour, gm.	Oxygen per Square Meter per Hour gm.	Respiratory Quotient
S	174	5.010	0.3505	12.27	10.56	0.847
	180	5.115	0.3553	12.22	10.81	0.824
P	149	4.220	0.3124	12.35	10.52	0.856
	169	4.430	0.3228	12.64	11.08	0.832
L	87	4.980	0.3491	12.33	12.22	0.730
	93	5.040	0.3519	11.48	10.93	0.760

results found in this observation with those secured with other infants in the previous work done by them, and find an increase in the carbon dioxid output of 21 per cent. over the results obtained with the breast-fed infant previously studied. (See Table 6.) This increase they explain by saying that it is due to the greater activity of the infant in the last experiment.

In 1908 a report appeared of the first in a remarkable series of experiments carried out by Schlossmann and Murschhauser in Düsseldorf.[23] The protocols of this experiment were given in connection with a description of the testing of the modified Regnault-Reiset apparatus constructed by Zuntz and Oppenheimer. The authors, Schlossmann,

22. Rubner and Heubner: Ztschr. f. exper. Path. u. Therap., 1904-05, i, 1.
23. Schlossmann, Oppenheimer and Murschhauser: Biochem. Ztschr., 1908, xiv, 385.

Oppenheimer and Murschhauser, emphasize the importance of observations when the infant is asleep; they accordingly preferred to make their observations the first half of the night, feeding the infant with a large amount of breast-milk in the early evening. The measurements of the metabolism of the infant during this experiment are given in Table 7. During the experimental period the infant weighed 5.79 kg., the calculated body-surface[24] being 0.384 square meters.

The same infant was subsequently studied by Schlossmann and Murschhauser[25] at the ages of 144 days, 284 days and 380 days. They found no difference in the metabolism per square meter of body-surface and conclude that Rubner's law is correct and that the metabolic processes are proportional to the body-surface.

The report of the first extensive research made by Schlossmann and Murschhauser appeared in 1910.[26] This is of special interest, inasmuch as the authors recognize at the outset the importance of muscular repose and of determining the basal metabolism. Many valuable suggestions as to the selection of infants for such study are incorporated in the report. Observations were made on three female infants; the results of these are given in Table 8. The authors conclude that the carbon dioxid production and the oxygen consumption depend on the body-surface, being wholly independent of the age of the subject, and draw the general conclusion that the infant produces per square meter of body-surface about 12 gm. of carbon dioxid and consumes about 11 gm. of oxygen per hour.

Recognizing the possibility that the environmental temperature may have an effect on the metabolism of the infant, Schlossmann and Murschhauser discussed this point in 1911,[27] giving the results of experiments with an infant in which the temperature of the surrounding atmosphere varied from 16.3 C. to 23.4 C. Another infant was studied with temperatures varying from 16.9 C. to 23.4 C. The results of this second experiment substantiated those obtained with the first infant, and the authors feel justified in concluding that the slight temperature changes found in experiments with an apparatus of the Regnault-Reiset type are entirely without influence on the metabolism of the individuals studied.

24. Surface $= 11.9 \sqrt[3/2]{w}$.
25. Schlossmann and Murschhauser: Biochem. Ztschr., 1909, xviii, 499.
26. Schlossmann and Murschhauser: Biochem. Ztschr., 1910, xxvi, 14.
27. Schlossmann and Murschhauser: Biochem. Ztschr., 1911, xxxvii, 1.

The same investigators,[28] with a keen appreciation of the influence of muscular activity on metabolism, compared the results of observations with an infant who cried continuously for an hour with those obtained when the infant was approximately quiet. They estimated that the crying increased the carbon dioxid production 59.4 per cent. and the oxygen consumption 44 per cent.

In still another paper Schlossmann[29] discusses the general principles involved in the measurement of the respiratory exchange of infants and emphasizes the necessity of muscular repose and the absence of food, and the importance of measuring the basal metabolism. He again asserts that the heat per square meter of body-surface is constant and maintains that this is a proof that the metabolism of young individuals is not variable. In this paper, also, he discusses the amount of work the infant does, basing the discussion on results obtained in his experiments with crying infants. In many of his experiments Schlossmann measured the skin temperature of the infant by electrical methods and found that there was no increase in the temperature.

In a paper discussing his earlier experiments on infants of varying ages and particularly those with an atrophic infant, Schlossmann opposes the views defended by Kassowitz,[30] that the metabolism in smaller animals is more intense than in the large animals and that there is no relationship between the metabolism and the body-surface.[31] Schlossmann maintains that atrophic infants have a higher metabolism per unit of body-surface than normal infants, but that this points to the correctness of the Rubner law, since with these infants the relation between body-surface and body-weight is abnormal. As he made no measurements of the body-surface of these infants—a procedure that necessitated an enormous amount of work—no direct evidence is offered to show that there was an actual disproportion between the body-surface and the body weight.

In a paper which appeared after the publication of Schlossmann's criticism, Kassowitz[32] sums up his arguments against the belief that the metabolism is proportional to the body-surface, and using Schlossmann's own protocols, criticizes adversely the latter's deductions.

28. Schlossmann and Murschhauser: Biochem. Ztschr., 1911. xxxvii, 23.
29. Schlossmann: Deutsch. med. Wchnschr., 1911, xxxvii, 1633.
30. Kassowitz: Allgemeine Biologie, Vienna, 1904, iii, 221.
31. Schlossmann: Ztschr. f. Kinderh., 1912-13, v, 227.
32. Kassowitz: Ztschr. f. Kinderh., 1913, vi, 240.

In a paper which has only recently appeared[33] Schlossmann again discusses the degree of activity and the amount of work done by infants in crying. He strongly emphasizes the necessity of noting the degree of repose during the observation, either by the ocular method used by himself or the graphic record devised in this laboratory. Unfortunately, at the time this paper was written, Schlossmann had not been able to compare the two methods.

TABLE 9.—RESULTS OF AN EXPERIMENT WITH AN ATROPHIC INFANT (NIEMANN)

Day	Carbon-Dioxid Production Per Day gm.	Average Temperature of the Air, °C.
1	108.0	20.5
2	110.4	20.0
3	117.6	21.0
4	115.2	21.0
5	120.0	21.0
6	127.2	21.0
Maximum	127.2	
Minimum	108.0	
Average	116.4	

TABLE 10.—AVERAGE CARBON-DIOXID EXCRETION IN EXPERIMENTS WITH AN ATROPHIC INFANT (FRANK AND WOLFF)

	Period I	Period II
Total twenty-four hours	127.61	148.81
Per hour	5.317	6.151
Per kilo per twenty-four hours	34.44	38.95
Per kilo per hour	1.435	1.623
Per square meter per hour	18.76	21.26

Two later communications by Schlossmann and Murschhauser[34] on the metabolism of fasting infants have particular significance in

33. Schlossmann: Monatschr. f. Kinderh., 1913, xii, 47. See also AM. JOUR. DIS. CHILD., 1913, vi, 15.

34. Schlossmann and Murschhauser: Biochem. Ztschr., 1913, lvi, 355; ibid., 1914, lviii, 483.

connection with this report, as they discuss the ideal conditions for obtaining the basal metabolism.

Using a Pettenkofer-Voit respiration apparatus[35] in the Kaiserin Auguste Victoria-Haus in Charlottenburg, Birk and Edelstein[36] studied the respiratory exchange of a healthy, new-born infant weighing 3.2 kg. and having a length of 50 cm. The infant was completely wound in cotton wool so as to keep the body-temperature at a normal level. Although he was removed from the respiration chamber several times during the day, the infant remained in the apparatus for the greater part of the twenty-four hours. On the second day the carbon dioxid production per twenty-four hours was 55.6 gm., or 18.2 gm. per kilogram; on the third day the total amount was 47.59 gm., or 15.76 gm. per kilogram per twenty-four hours. The authors criticize the use of short experiments with a respiration apparatus by which the oxygen consumption can be determined and the respiratory quotients calculated, but express regret that with their method the oxygen consumption cannot be determined.

In a study made by Carpenter and Murlin[37] of the energy metabolism of pregnant women before and after the birth of the child, the energy metabolism of three new-born infants was also found per unit of weight and per unit of body-surface. The values were obtained by subtracting the measured metabolism of the mother from that of the mother and infant together.

The metabolism of an atrophic infant was studied by Niemann,[38] who used a Pettenkofer-Voit respiration apparatus in the children's clinic of the University of Berlin. The observation continued six days, the infant remaining in the chamber the greater part of each day. The measurements of the carbon dioxid production on the basis of twenty-four hours are given in Table 9. When these results are computed on the basis of carbon dioxid produced per square meter of body-surface, using the formula of Meeh and the constant 11.9, the author finds that this infant with an average body-weight during the six days of 3.28 kg. had a body-surface corresponding to 0.2626 square meter, and that the

35. Bahrdt and Edelstein: Jahrb. f. Kinderh., 1910, lxxii, 43.

36. Birk and Edelstein: Monatschr. f. Kinderh., 1910, ix, 505.

37. Carpenter, T. M., and Murlin, J. R.: The Energy Metabolism of Mother and Child Just Before and Just After Birth, Arch. Int. Med., 1911, vii, 184.

38. Niemann: Ztschr. f. Kinderh., 1913, vi, 375.

carbon dioxid excretion per square meter of body-surface was 18.5 gm. per hour.

The metabolism of another atrophic infant was studied in the Universitäts-Kinderklinik, Berlin, by Frank and Wolff.[39] Using the Pettenkofer-Voit respiration apparatus modified by Rubner, they made two series of experiments of four days each. The average values for carbon dioxid are given in Table 10. The authors especially comment on the unusually high figures for the carbon dioxid excretion, and attempt to explain them by the disturbance between the computed body-surface and body-weight and the effect of a protein-rich diet, but expressly maintain that muscular activity played no rôle as the infant, except on the first day, was *sehr ruhig*.

Bahrdt and Edelstein[40] also report an experiment with an atrophic infant, in which they used the respiration apparatus in Langstein's laboratory in the Kaiserin Auguste Victoria-Haus. The observation was made in three periods of four days each. In the first and last periods, the infant remained inside the chamber for the greater part of the twenty-four hours, being removed periodically, as is customary in experiments with this type of apparatus. Their final conclusion was that the heat-production of atrophic infants can be abnormally high aside from any effect which the environmental temperature or the body activity may have on it.

Finally, the direct calorimetric and gasometric researches of Howland[41] in Lusk's laboratory should be especially noted. In discussing the calculation of the body-surface, Howland cites the use of the factor 12.3 as a constant for infants, but we are not aware of any writers who have previously used this factor. The experiments were made with the respiration calorimeter[42] at the Cornell University Medical College in New York. Three infants under 1 year of age were studied and ocular observations of the activity of the infants were recorded.

Howland's experiments were subsequently published in detail and the results more fully discussed.[43] In this paper the relationship

39. Frank and Wolff: Jahrb. f. Kinderh., 1913, lxxviii, 1.
40. Bahrdt and Edelstein: Festschrift Dr. Otto L. Heubner, 1913, Berlin.
41. Howland: Proc. Soc. Exper. Biol. and Med., 1911, viii, 63; (Hoppe-Seyler's) Ztschr. f. physiol. Chem., 1911, lxxiv, 1.
42. Williams: Jour. Biol. Chem., 1912, xii, 317.
43. Howland: Tr. Fifteenth Internat. Cong. Hyg. and Demog., 1912, ii, 438. (Pub. Washington, 1913.)

between body-surface and body-weight is extensively treated and the various factors and formulas are considered. A curve is proposed, which is represented by the algebraic expression $y = mx + b$, in which y is the surface area of the infant, x the weight of the infant in grams, m the constant 0.483, and b, 730 sq. cm.

The last portion of the paper, which is of most significance, compares the direct and indirect computation of the heat-production of the infants observed. This comparison is of such value to workers in metabolism that it is given here in Table 11. The agreement between the heat-production as directly measured and as indirectly computed is striking, to say the least, and justifies for the present the utilization of the indirect method of computing the calorimetry of infants.

Though Howland, using Lusk's calorimeter, has been eminently successful in experiments on the direct calorimetry of infants, experience with such researches in the Nutrition Laboratory has led us to believe that a type of calorimeter with less mass, less heat capacity, and probably not of the continuous-flow type could most advantageously be employed for the short periods necessitated by experiments with infants. Several types or modifications of calorimeters have been in process of testing for some time, and pending the satisfactory development, construction and testing of an infant calorimeter with the qualifications referred to, we have actively undertaken the study of infant metabolism as determined by indirect calorimetry. In these observations we have devoted our energies to the accurate measurement, in relatively short periods, of the carbon dioxid produced and oxygen consumed by infants less than 1 year of age.

INDIRECT CALORIMETRY

A number of physiologists have used the respiratory exchange to compute the total calorimetry by the method of so-called "indirect calorimetry."[44] From analyses of the urine and from a careful study of the respiratory exchange it is possible to apportion the catabolism between protein, fat and carbohydrate according to methods worked out most carefully by Zuntz and his associates. Having apportioned this catabolism, it is relatively simple to compute the total caloric out-

44. This method is not to be confused with the usage of certain French writers who consider "indirect calorimetry" as indicating the computation of material consumed by noting the weights of food eaten, excreta, and gain or loss of body-weight.

put from the well-known heat of combustion of the various nutrients. Thus, for every gram of nitrogen in the urine, it has been definitely shown that 26.51 calories are developed; for every gram of carbohydrate burned in the body, not far from 4 calories are produced, and for every gram of fat, 9.5 calories.[45]

A simpler method of computing the total heat, however, is to utilize the respiratory quotients, that is, the ratio between the carbon dioxid produced and the oxygen burned. Accordingly, if one knows exactly the respiratory quotient, the total carbon dioxid produced, and the

TABLE 11.—HEAT-PRODUCTION OF INFANTS, DIRECTLY AND INDIRECTLY MEASURED, AS REPORTED BY HOWLAND

Subject	Food	Calories per Sq. M. per Day		Difference Per Cent.
		Measured	Calculated	
Child 1.....	Ordinary	1,046	1,084	2
		1,113	1,174	
		1,196	1,164	
Child 1.....	Nutrose added	1,218	1,179	3
Child 1.....	Nutrose added	1,204	1,180	1—
		1,235	1,212	
		1,181	1,250	
Child 1.....	Fasting	1,106	1,177	1—
		1,226	1,156	
		1,301	1,243	
Child 3.....	Ordinary	858	793	2
		915	933	2
	Ordinary	825	840	

oxygen absorbed, it is relatively simple to compute the total energy by using the so-called calorific equivalent for either carbon dioxid or oxygen. These calorific equivalents have also been most carefully studied by Zuntz and his associates, and as a result we have a very helpful table which gives the calorific equivalent of carbon dioxid with various respiratory quotients. When pure fat is burned, the respiratory quotient is not far from 0.71, and Zuntz has shown that for every liter of carbon dioxid produced by burning pure fat, 6.606 calories are liberated, or 3.363 calories per gram of carbon dioxid. On the other hand, when pure carbohydrates are burned, the ratio between carbon dioxid and oxygen is 1.00, that is, for every liter of carbon dioxid

45. The heat of combustion of anhydrous cow's milk fat is 9.2 calories.

produced, 1 liter of oxygen is absorbed. Under these conditions Zuntz has shown that each liter of oxygen consumed corresponds to 5.047 calories, or 2.569 calories per gram of carbon dioxid.

An extremely simple method for calculating the catabolism, therefore, is to multiply the total amount of carbon or oxygen measured by the corresponding calorific equivalent. As a matter of fact, Zuntz in his work has used most frequently the calorific equivalent of oxygen, for in his method of studying the respiratory exchange the determinations of oxygen are somewhat more exact than are those of carbon dioxid. Since, in our respiration apparatus, the carbon dioxid deter-

TABLE 12.—CALORIFIC EQUIVALENTS OF CARBON DIOXID

Respiratory Quotient	Calorific Value of Carbon Dioxid		Respiratory Quotient	Calorific Value of Carbon Dioxid		Respiratory Quotient	Calorific Value of Carbon Dioxid	
	Per Liter cals.	Per gm. cals.		Per Liter cals.	Per gm. cals.		Per Liter cals.	Per gm. cals.
0.70	6.694	3.408	0.80	6.001	3.055	0.90	5.471	2.785
0.71	6.606	3.363	0.81	5.942	3.025	0.91	5.424	2.761
0.72	6.531	3.325	0.82	5.884	2.996	0.92	5.378	2.738
0.73	6.458	3.288	0.83	5.829	2,967	0.93	5.333	2.715
0.74	6.388	3.252	0.84	5.774	2.939	0.94	5.290	2.693
0.75	6.319	3.217	0.85	5.721	2.912	0.95	5.247	2.671
0.76	6.253	3.183	0.86	5.669	2.886	0.96	5.205	2.650
0.77	6.187	3.150	0.87	5.617	2.860	0.97	5.165	2.629
0.78	6.123	3.117	0.88	5.568	2.835	0.98	5.124	2.609
0.79	6.062	3.086	0.89	5.519	2.810	0.99	5.085	2.589
						1.00	5.047	2.569

minations for short periods are even more exact than are the determinations of the oxygen, we give in Table 12 the calorific equivalents of carbon dioxid with the varying respiratory quotients, particularly as this table will be used extensively in the computation of our own researches.

As any form of indirect calorimetry must of necessity be somewhat speculative,* one must always rely for fundamental values on direct

* It will be noted that in this paper the computation of the energy derived from protein is neglected and that the total energy output is computed only from the amounts of carbon dioxid and oxygen. The possible error in neglecting the protein has been computed by Magnus Levy to be somewhat under 1 per cent., and as the determinations of nitrogen were not feasible in our studies, we have used the method of simple computation from the gaseous exchange as recommended by A. Loewy: Oppenheimer's Handbuch der Biochemie, Jena, 1911, iv, 281. See, also, Magnus-Levy: von Noorden's Handbuch der Pathologie des Stoffwechsels, Berlin, 1896, i, 207.

heat measurements. Such measurements have been extensively made with men by Atwater and his associates at Wesleyan University, Middletown, Conn., where it was shown in experiments of long duration that the heat output as measured directly by the respiration calorimeter and the heat output as computed from the respiratory exchange agreed remarkably well. It should be pointed out, however, that these computations were based on twenty-four-hour periods. In certain experiments the computation has likewise been successful for periods as short as six hours, but it remained for Howland[46] to demonstrate with Lusk's calorimeter the complete agreement of the direct calorimetric measurements and of the computation from the gaseous exchange for short periods and particularly with an infant as subject. This being the case, the field is open for making a large number of metabolism experiments with the respiration apparatus in laboratories and institutions where a respiration calorimeter for direct calorimetry is not available.

BASAL METABOLISM

While the normal life of the infant is a relatively simple and constant one, yet it does include periods of muscular activity which vary greatly, the extremes ranging from perfect muscular repose during sleep to the violent exercise incidental to vigorous crying. As a result of these changes in muscular activity, the output of heat would likewise vary largely, with a minimum output during sleep and quiet and a maximum during the period of crying. An attempt has been made to find the average value for the heat output of an infant by experiments with twenty-four-hour periods, thus securing an average for the life of the day; but when one considers that the difference between the heat output in complete muscular repose during sleep and the heat given off when the infant is crying vigorously may be as great as 60 or more per cent., it will be seen that this method of averaging does not furnish information with regard to the minimum metabolism.

The muscular activity of infants is twofold: (1) internal muscular activity, consisting of muscular tonus, the movements of the organs of circulation and respiration, and possibly the processes of digestion; and (2) external muscular activity such as the movements of the

46. Howland: Tr. Fifteenth Internat. Cong. Hyg. and Demog., Washington, 1913, ii, 451.

limbs or vigorous body movement incidental to crying. The internal movements can be controlled only by minimizing the after-effects of digestion through the absence of food; the regular involuntary muscular activity of the respiratory and circulatory system and the muscular tonus cannot be altered. On the other hand, the external muscular activities are at a minimum only during complete muscular repose, as in deep sleep. It is thus seen that the ideal conditions for studying the basal or minimum metabolism of infants would be the postabsorptive state, that is, sufficiently long after the last meal to insure that there was no longer an absorption of food materials from the alimentary tract, and during deep sleep when there is complete muscular repose. With very young infants, periods of complete muscular repose cannot be expected for any great length of time, probably not for more than two successive hours.

We have, then, two factors to deal with: first, the heat elimination incidental to the specific catabolic stimuli of the food materials accompany the digestion and absorption of food, and second, the internal muscular activity of the infant. If the first of these factors can be eliminated, we have what may properly be termed the basal metabolism unaccompanied by extraneous muscular activity of any kind.

Accordingly, in our first years of experimenting, we have based our study of the metabolism of infants on the careful determination of the carbon dioxid production and oxygen consumption of infants under one year in a special form of respiration apparatus, under conditions favoring the accumulation of accurate information regarding the basal metabolism.

RESPIRATION APPARATUS

The apparatus used in this research was a modified form of that briefly described by us in our first communication.[47] The essential modifications are, first, arrangements for continuous observations whereby periods of any desired length can be secured, and, second and most important, the method of determining the oxygen consumption directly. The apparatus has been carefully controlled by burning known weights of alcohol, securing not only the respiratory quotients, but likewise the total amount of carbon dioxid and oxygen corresponding to the given amount of alcohol burned. For a complete description

47. Benedict, F. G., and Talbot, F. B.: Some Fundamental Principles in Studying Infant Metabolism, AM. JOUR. CHILD. DIS., 1912, iv, 130.

of this apparatus and tests made with it, reference may be made to the large publication previously referred to.[48] As the infant gives off carbon dioxid and consumes oxygen, the air leaving the chamber is rich in carbon dioxid and water-vapor from the lungs and skin of the infant, contains a normal amount of nitrogen, and is deficient in oxygen. By means of a rotary pump, the air is carried from the chamber and forced through sulphuric acid which absorbs the water, then

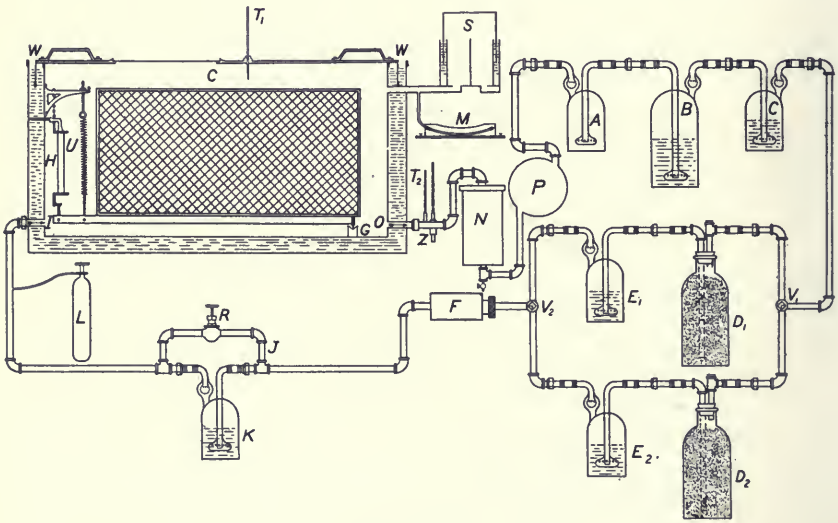

Fig. 1.—Detailed scheme of respiration apparatus. *C*, chamber; *W*, *W*, water jacket; *O*, outgoing air-pipe; *Z*, psychrometer; *N*, muffler; *P*, blower; *A*, acid trap; *B* and *C*, Williams water-absorbers; V_1 and V_2, 2-way valves; D_1 and D_2, carbon dioxid absorbers; E_1 and E_2, air-dryers; *F*, sodium bicarbonate can; *J*, by-pass; *R*, valve; *K*, air moistener; *L*, oxygen cylinder; *I*, ingoing air-pipe; *S*, spirometer; T_1 and T_2, thermometers; *M*, manometer; *U*, spiral spring; *H*, pneumograph.

through soda lime to remove the carbon dioxid; oxygen is next introduced and when the air returns to the chamber it is free from carbon dioxid and water and contains a normal percentage of nitrogen and oxygen. A scheme of this respiration apparatus, giving in considerable detail some of the special connections, may be seen in Figure 1.

48. Benedict, F. G., and Talbot, F. B.: Carnegie Institution of Washington, Publication No. 201, 1914.

METHOD OF RECORDING THE DEGREE OF MUSCULAR REPOSE

Special care was taken to secure graphic records of the degree of muscular repose of the infant, and each experiment in the entire series of some 1,200 periods, with about eighty infants, was accompanied by a graphic registration of the degree of muscular repose. This was accomplished by resting one end of the crib on a knife-edge bearing and suspending the other on a spiral spring, so that even the slight changes in the center of gravity of the infant would alter the tension on the spring. One end of a pneumograph was attached to the crib and the other end to the wall of the chamber, the pneumograph thus being parallel to the spiral spring. The slightest lengthening or shortening of the pneumograph produced a change in the tension of the confined air, these varying air tensions being transmitted by a tube through the walls of the chamber to a delicate tambour and pointer which gave graphic records on a kymograph drum. It is only necessary here to give one characteristic tracing, illustrating the records obtained by this method as to the degree of muscular repose. This curve, which is shown in Figure 2, was obtained Jan. 28, 1914, with G. M., an infant weighing 3.73 kg.

At the bottom of the figure is shown the record of the sensitivity test which precedes each experiment. In these tests, weights equal to the weight of the infant are placed in the crib; a 50-gm. weight is then dropped from a height of 21 cm. so as to strike the crib a blow at approximately 32 cm. from the knife-edge bearing on which the crib rests, thus giving an impulse to the crib, the vibrations gradually slowing down. Tests are made with the kymograph set first at slow speed and then at fast speed. In the record of the experimental period, the major movements are shown by the larger amplitude. It will be seen that the infant quieted down sufficiently to begin the first period of the experiment at 3:05 p. m. Aside from the slight movements at the end of the period, the infant was very quiet until 3:28 p. m. In the period from 3:28 p. m. to 3:59 p. m., there were several major movements and toward the end of the next period the infant was obviously very restless. This curve is of particular interest in that it shows in the period from 4:23 p. m. to 4:50 p. m. the movements of the crib incidental to hiccoughs. Indeed, it is possible so to adjust the sensitivity of the crib as to record the respirations.[49]

49. Benedict, F. G., and Talbot, F. B.: Carnegie Institution of Washington, Publication 201, 1914, p. 58 (Fig. 6).

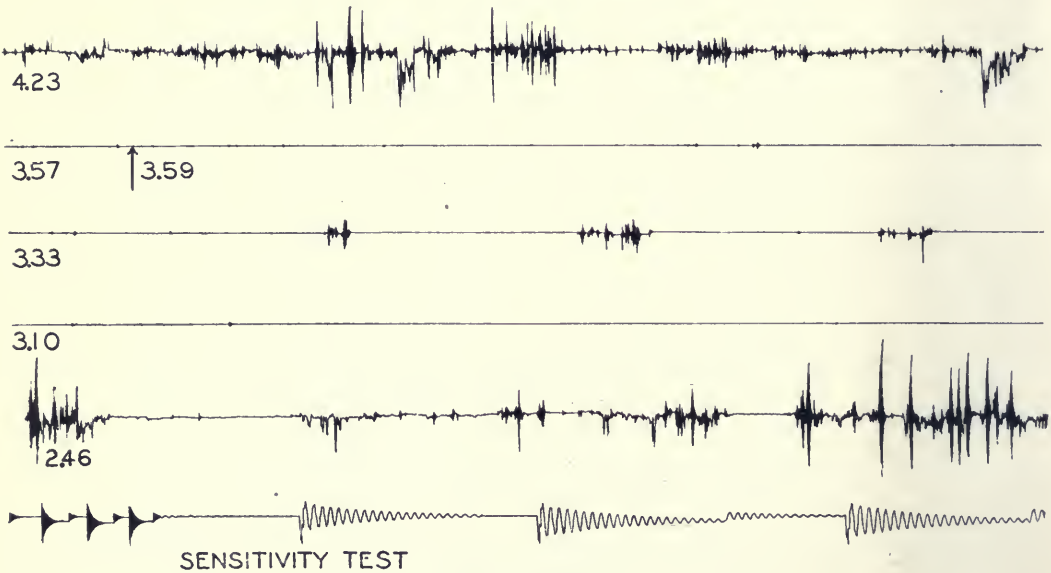

Figure 2—Part 1.

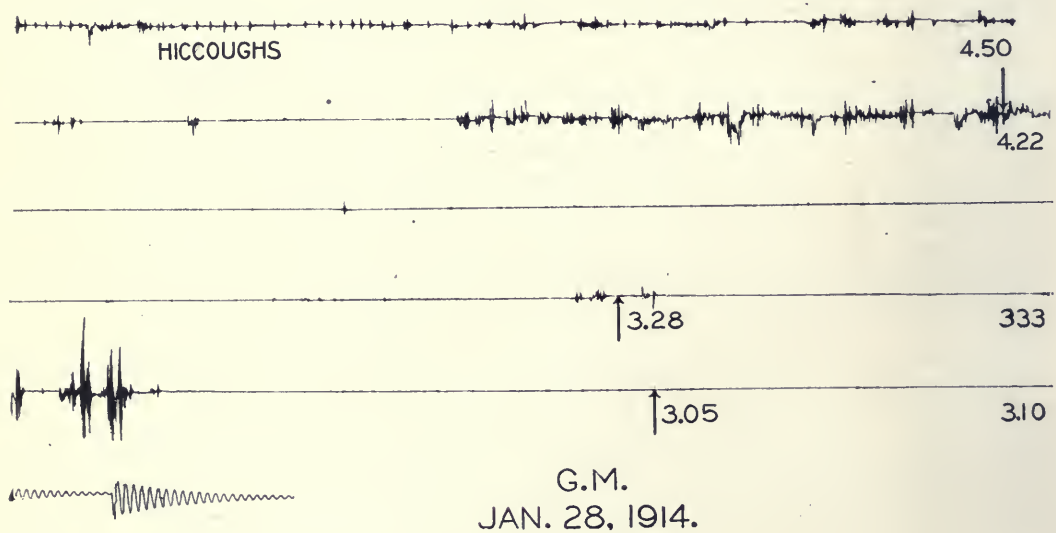

Figure 2—Part 2.

Fig. 2.—Typical kymograph curve, showing sensitivity test and records of muscular activity.

At first the infant was observed through the window in the cover of the apparatus to see whether or not he was quiet. While the ocular method of recording changes in position and the activity of an infant is much to be preferred to the terse statements usually accompanying reports of metabolism experiments to the effect that the animal, infant or person was "quiet," "fairly quiet" or "restless," yet such observations cannot be relied on. We have repeatedly seen experimental periods when a careful observer, even though watching the infant continuously, was unable to record a perceptible movement other than those of respiration, and yet the suspended crib, pneumograph and tambour have recorded distinct and persistent muscular tremors, accompanied in all cases by an increasing pulse-rate and increased metabolism as measured by the carbon dioxid production and oxygen consumption. The visual estimation was therefore discontinued as being too inaccurate and unreliable a record of the degree of quiet.

The length of a period of observation depends altogether on the muscular repose of the infant, as only quiet periods, accompanied by a low pulse-rate, are of value. With a small, quiet infant, the periods may vary in length from twenty to thirty minutes, but with a large infant they may be as short as fifteen minutes.

The respiration apparatus as installed in the Children's Department of the Massachusetts General Hospital is shown in Figure 3. It consists of the respiration chamber, with oxygen supply, meter, balances for weighing, air-purifying apparatus, barometer and tambour and kymograph for recording the degree of muscular repose.

WARD CRIB RECORDER

The relationship observed between the graphic tracings of the muscular activity and the catabolism indicated the possible value of recording the activity of the infant throughout the day when it was not inside the respiration chamber. Accordingly, to assist in settling some complicated problems of nutrition, a special apparatus, embodying the suspended crib principle, was devised and set up in the children's ward of the hospital in order to obtain a continuous graphic record of the muscular activity of the infant (Fig. 4).

In this apparatus the wire crib rests at the rear end on two steel points fitting into steel cups. The front end is suspended by a spring with link chain adjustment for height. At the right of the front end

Fig. 3.—General view of the infant respiration apparatus. In the foreground is the respiration chamber showing the crib inside. The cover of the chamber rests on the floor, and through the glass window may be seen the earpieces of the stethoscope used for recording the pulse-rate. On the shelf in front of the chamber are the tambour and kymograph for the graphic records of movements. Directly behind the chamber are the hygrometer, the gas meter immersed in water, and the cylinder of oxygen, while in the rear the spirometer may be seen at the right and the sulphuric acid and soda lime containers of the absorbing system at the left. In the corner of the room is the balance for weighing the absorbing system.

is an aluminum wheel attached to a revolution counter. A fine thread fits in a groove on the edge of this wheel. One end of this thread is attached to the upper end of the cage and the other end to a fine spiral spring, projecting to the right of the revolution counter. Each movement of the cage in a vertical direction moves the revolution counter, and as the cage descends, the fine spring keeps the tension on the thread tight. The pawl on the top prevents backward motion of the wheel and the pawl-standard serves as a pointer to read fractions of the circumference of the wheel.

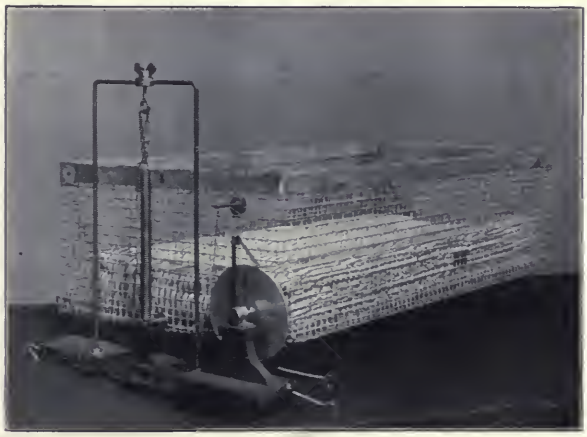

Fig. 4.—Ward crib recorder.

METHOD OF RECORDING THE PULSE-RATE

Previous experiments with adults in the Nutrition Laboratory, in which the large respiration chambers were used, showed a striking relationship between the pulse-rate and the metabolism. Attempts were accordingly made to secure accurate pulse-records in our observations with infants. For this purpose we attached the bell of a small Bowles stethoscope to the infant over the apex-beat of the heart by means of strips of adhesive plaster. A rubber tube connecting with the bell led to a pipe in the wall of the chamber, a piece of rubber tubing and earpieces being attached to the outer end of the tube. Even with a total length of some 2 or 3 meters from the bell to the earpieces, it was possible to count the pulse-rate of the weakest infant. Throughout

TABLE 13.—CLINICAL STATUS OF INFANTS STUDIED

Name	Age During Metabolism Observation	Clinical Status
F. B.	5½ mos...	Convalescent stage infantile atrophy.
M. A.	9 mos...	Under weight, splenic tumor, with anemia.
J. B.	5 mos...	Congenital syphilis, convalescent stage infantile atrophy.
L. B.	4 mos...	Under weight.
L. R. B.	4–4½ mos...	Normal infant.
A. C.	1½ mos...	Slightly under weight.
M. C.	4 mos...	Normal infant or slightly under weight.
A. D.	4–5 mos...	Convalescent stage, infantile atrophy.
M. D.	2–3 wks...	Normal infant.
R. E.	4½ mos...	Under weight or slightly under expected weight.
E. F.	3 mos...	Normal infant.
E. G.	10 mos...	Normal infant.
E. K.	17 mos...	Much under weight; rachitis, recovering from broncho-pneumonia.
F. K.	7 mos...	Under weight.
A. L.	3½–4 mos...	Under weight.
E. L.	4 mos...	Under weight, otitis media.
R. L.	6½–9 mos...	Approximately normal; later, under weight.
D. M.	11 mos...	Much under weight, rachitis.
F. M.	4–5 mos...	Under weight, congenital syphilis.
J. M.	8 mos...	Under weight, otitis media, rachitis.
M. M.	4½ mos...	Under weight following an acute indigestion.
E. N.	6–6½ mos...	Under weight.
L. O.	5–6 mos...	Infantile atrophy.
J. P.	6½–7 mos...	Under weight, gaining weight rapidly.
W. P.	5–5½ mos...	Under weight.
D. Q.	4½ mos...	Under weight.
E. R.	3 mos...	Under weight (slightly).
K. R.	4 mos...	Infantile atrophy.
A. S.	3 mos...	Normal infant, weighing more than the average.
E. S.	5 mos...	Infantile atrophy.
E. H. S.	2½–4 mos...	Infantile atrophy.
G. S.	2½ mos...	Under weight.
J. S.	5–6 mos...	Infantile atrophy (?) (temperature not subnormal).
P. S.	12 mos...	Under weight.
H. T.	5½ mos...	Normal infant, weighing more than average.
J. V.	3½–9 mos...	Prematurity; congenital syphilis; infantile atrophy in subnormal temperature and convalescent stage.
P. W.	7 mos...	Normal infant.
New Series		
R. A.	7 mos...	Moderately under weight.
E. C.	2½ mos...	Moderately under weight with indigestion.
H. C.	5 mos...	Much under weight.
O. C.*	5 hrs.–3 das.	Normal infant.
R. C.*	1–2 days...	Normal infant.
T. C.	8½–9 mos...	Much under weight.
B. D.	2 mos...	Normal infant. Breast fed.

TABLE 13.—CLINICAL STATUS OF INFANTS STUDIED—(*Continued*)

Name	Age During Metabolism Observation	Clinical Status
F. D.	2½ mos...	Congenital syphilis. Normal weight.
N. D.	7 mos...	Moderately under weight.
R. D.*	2–5 days..	Normal infant of average weight.
B. F.	7 mos...	Moderately under weight.
U. H.*	3–28 hours.	Normal infant.
T. K.*	7–32 hours.	Normal infant.
L. L.	2–2½ mos...	Normal infant.
E. M.	8 mos...	Moderately under weight.
G. M.	6–6½ mos...	Much under weight. Eczema. Indigestion.
C. N.	4½ mos...	Eczema. Much under weight with indigestion.
I. N.	2–4 days..	Normal infant.
J. O.	4½ mos...	Pyloric spasm. Indigestion. Much under weight.
E. P.	11–14 days..	Normal infant.
F. R.	1–11 days..	Normal infant. Much above average weight.
I. R.	6–7 days..	Normal infant.
R. S.	9½ mos...	Normal infant. Much over average weight.

*These infants were obtained through the courtesy of the visiting physician of the Boston Lying-In Hospital, Dr. Charles Montraville Green.

the entire period of observation, the pulse-rate was recorded every two minutes by an assistant who made this her sole duty.

We are far from satisfied with this as a permanent method for securing a record of the pulse-rate, and it is our hope, in connection with the hospital or with the laboratory, to secure records either with the string galvanometer or with the Bock-Thoma oscillograph. It is clear that the records of the pulse-rate should be more objective than they can be even with a specially detailed assistant.

SELECTION OF SUBJECTS

The infants studied were either from the Children's Department of the Massachusetts General Hospital or from the Boston Lying-In Hospital. The routine histories, records of the physical examinations, notes regarding the urine, stools, blood and temperature, the pulse and respiration charts, and detailed records of the food were kept for all of the infants. The Wassermann reaction and the von Pirquet skin tests were also made in many cases. It does not seem desirable to publish the detailed hospital record of each infant that came under observation, especially since most of the evidence is negative as to whether or not

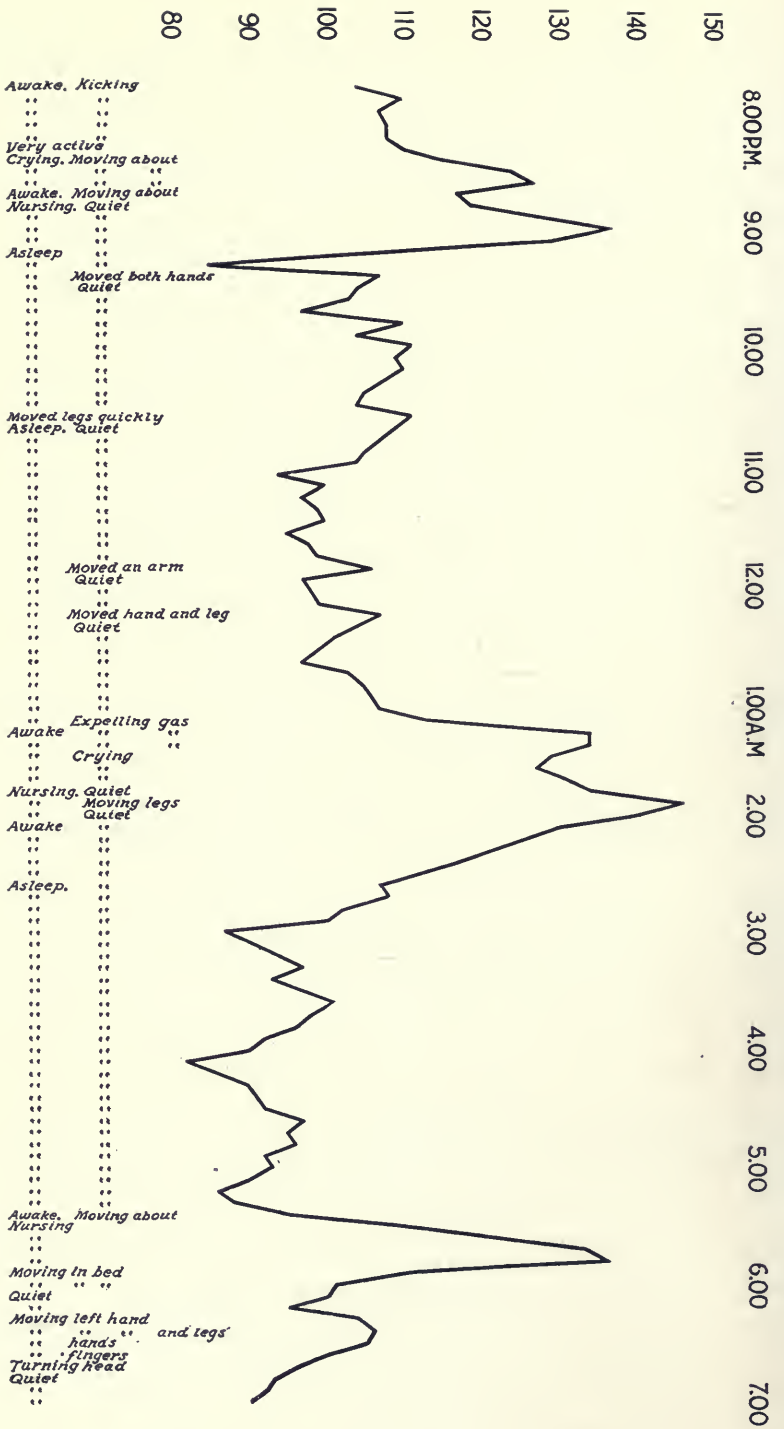

Fig. 5.—Pulse-rate curve for Tremballe, July 12, 1911. Age, 5 months; weight, 5.7 kg.

an infant is normal. The clinical status of each infant is, however, given in Table 13.

In this table the term "infantile atrophy" is applied to the condition of an emaciated infant with such severe indigestion that it is unable to digest weak mixtures of cow's milk, with no gain in weight, and with a subnormal body-temperature. The convalescent stage of infantile atrophy is that in which the same infant subsequently begins to digest its food and to gain weight and has a normal temperature. Underweight infants are those who are 0.5 kg. or more below the average weight for their respective ages, but whose digestion is not so severely deranged as those with infantile atrophy. This group includes all infants not classified as normal, or with infantile atrophy, or in the convalescent stage of infantile atrophy.

DISCUSSION OF RESULTS

In all of the researches carried out in the Nutrition Laboratory, particularly in studying pathologic cases, the want of suitable and adequate control data for comparison has been evident. With infants, particularly, the absence of normal controls is unfortunate. This led us to believe that our best service to pediatrics would be to determine the normal metabolism of infants. Accordingly, while a few observations were made on distinctly pathologic cases other than atrophic, our data were secured for the most part with normal or underweight infants.

One of the most important problems in such an investigation is the relationship between muscular repose and metabolism. In our first communication to this journal, we emphasized the importance of this relationship and pointed out the great service rendered by the graphic registration device for recording the muscular movements of the infant. Our consistent use of this device in all of our experiments furnished us with data in regard to the relationship between muscular repose and metabolism, which was accordingly made the subject of special study in this investigation.

In this research, also, it was hoped that some information would be gained as to the cause for variations in the metabolism of the infant; with this in view, a study was made of the possible relationships between the metabolism and the body-surface and the metabolism and the active mass of protoplasmic tissue.

1. Pulse-Rate

The careful observations of pulse-rate made while the subjects were inside the respiration chamber verified completely the twelve-hour observations made in the ward and published in our earlier paper. These showed very considerable fluctuations in the pulse-rate coincident with changes in muscular activity, such as change in body position, particularly in crying and immediately after nursing, so far as the movements inside the respiration chamber approximated those noted in the ward. For an elaborate series of observations on the pulse-rate

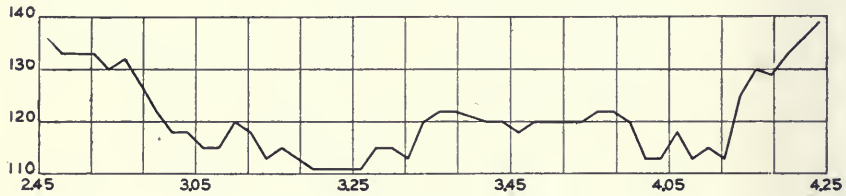

Figure 6a.

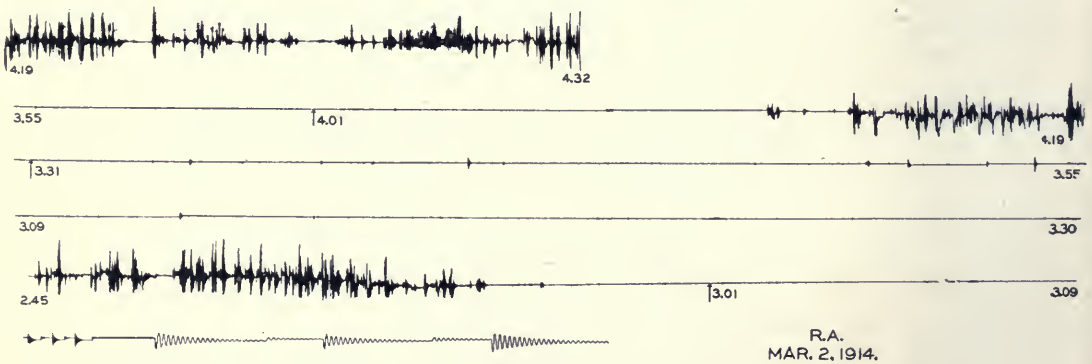

Figure 6b.

Fig. 6.—Pulse-rate and kymograph curves for R. A., March 2, 1914.

as affected by the muscular activity incidental to a night's sojourn in the wards, our earlier paper may be consulted, as well as a number of records given more recently in the Carnegie Institution of Washington publication.

The surprising changes in pulse-rate found in the course of an ordinary night's sleep in the ward are shown in Figure 5 by the typical curve for the 5-months-old infant, Tremballe. Accompanying the curve are statements with regard to the condition and activity of the

child. Tremballe was under observation in the ward from 7:40 p. m. until about 7 a. m., and for the greater part of this time was asleep. The pulse-rate varied from a maximum of 147 to a minimum of 83. During the latter part of the night the minimum record showed an average of not far from 90. An immediate and rapid effect on the pulse following changes in muscular activity is strikingly shown with this infant in every instance.

TABLE 14.—COMPARISON OF THE PULSE-RATE, METABOLISM, AND MUSCULAR ACTIVITY IN OBSERVATION WITH M. M., JUNE 5, 1913

Period	Total Heat Production per 24 Hours	Pulse-Rate	Activity
3:12 p. m. to 3:40 p. m.*...	365	107	V
3:40 p. m. to 4:10 p. m.....	276	93	I
4:10 p. m. to 4:31 p. m.....	307	96	III
4:31 p. m. to 4:58 p. m.....	288	90	II
4:58 p. m. to 5:23 p. m.....	367	113	VI

* Preliminary period.

2. Relationship Between Pulse-Rate and Muscular Activity.

While the observations made in the ward during the night experiments in 1911 showed conclusively the intimate relationship between pulse-rate and activity, this is even more strikingly brought out by the kymograph pictures obtained while the infant was inside the respiration chamber. With each change in bodily activity, there was obviously a noticeable change in the course of the kymograph record, and by means of frequent pulse-counts, we were able to make sharp comparisons between these two factors. A curve showing graphically the pulse-counts obtained in a typical experiment with R. A. on March 2, 1914, is given in Figure 6. For comparison, the kymograph record, somewhat reduced, is also given.

In comparing the two curves, we find that the period of considerable activity from 2:45 p. m. until about 2:58 p. m. is accompanied by a high pulse which fell rapidly as the infant quieted down. From 3:01 p. m. to 3:30 p. m., the kymograph curve shows a straight line, with but one or two slight movements. From 3:31 p. m. to 3:55 p. m., there is distinct evidence of slight restlessness, which is also apparent in the

increased pulse-rate, this period being followed by complete repose from 3:55 p. m. to about 4:10 p. m., and a lowering of the pulse-rate. From that time until the end of the experiment, there is great activity and disturbance, with a rapidly rising pulse-rate. It is thus seen that the kymograph curve and the pulse-rate curve follow each other with great regularity.

3. Relationship of the Muscular Activity, Pulse-Rate and Metabolism

The noticeable influence of even minor muscular activity on metabolism makes it important to note that in all experiments in which the basal metabolism is to be determined, that is, in experiments that

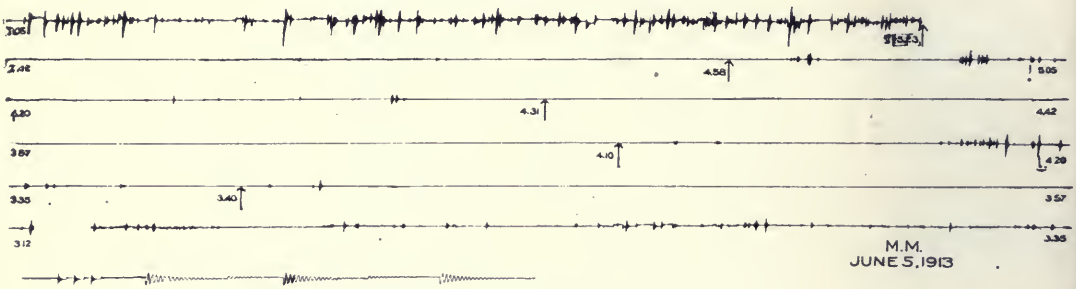

Fig. 7.—Kymograph curve for M. M., June 5, 1913.

may subsequently be employed for comparison of normal individuals with pathologic cases, only periods of complete muscular repose should be used. It is obviously impossible to compare a quiet, resting, normal child with a sick, restless child, or vice versa. The difficulties incidental to securing such periods are known only to those who are actively engaged in this sort of experimenting, for a very large percentage of the results of experimental work may not be used, owing to changes in the extraneous muscular activity. Writers have, as a rule, attempted to indicate in a general way the results of certain experiments which cannot properly be included in comparisons by recording in some general terms the degree of activity of the child, stating whether it is awake or asleep, or comparatively quiet, or quiet, etc. In recent years Schlossmann and Murschhauser have adopted a more elaborate system of ocular records, stating exactly what the

infant is doing. After comparing the various methods of recording the degree of muscular repose, we have definitely adopted the suspended crib and kymograph.

It is clearly impracticable to reproduce in this publication all of the kymograph curves secured in our researches, but a comparison may be made of the muscular activity, the pulse-rate, and the metabolism in a typical experiment—that with M. M. on June 5, 1913—by reference to Table 14. In this table are given a record of the pulse-rate, the metabolism computed on the basis of the total heat output for twenty-four hours, and an estimate of the activity based on the following classification:

I. Very quiet, probably asleep.
II. Slight movements, few in number.
III. Some activity, but generally quiet.
IV. Moderately active.
V. Distinctly active.
VI. Very active, most or all of the time.

The kymograph curve for the same experiment, which is reproduced in Figure 7, will give an illustration of the method of estimation we are using. This curve has certain striking points, inasmuch as the minimum and maximum activity are very well shown. During the preliminary period from 3:12 p. m. to 3:40 p. m., the infant was somewhat restless, quieting down sufficiently about 3:35 p. m. to justify the beginning of a new period at 3:40 p. m. As a matter of fact, the infant was so quiet in the next period that the activity can be characterized as I (Table 14). The activity in the last period, that is, that from 4:58 p. m. to 5:23 p. m., was sufficiently great to be classified as VI. This curve shows clearly the futility of attempting to graduate by kymograph records the exact degree of the activity and the heat-production, for although the curve appears to indicate that the activity in the last period (from 4:58 p. m. to 5:23 p. m.) was much greater than that in the preliminary period from 3:12 p. m. to 3:40 p. m., the metabolism is very nearly the same and the pulse-rate is only 6 beats higher in the last period. This also justifies the statement that the measurements obtained in preliminary periods are not sufficiently reliable to admit of extended discussion. The fact that no greater metabolism is shown in the last period than in the preliminary period, although the activity appears to be greater, should therefore be considered as a deduction based on single measure-

ments in two individual periods, either of which may be liable to error. Furthermore, when comparing the pulse-rates it should be stated that although the average pulse-rate in the preliminary period was 107, the individual counts ranged from 95 to 120, while the pulse-rates in the last period, although the average was 113, actually varied from 93 to 124. Discrepancies such as these serve again to emphasize the fact that only periods of complete muscular repose can logically be used in discussing infant metabolism.

4. Significance of the Relationship

From the preceding discussion the conclusion may be drawn that only periods of complete muscular repose may be used in comparing the results obtained with different individuals and with the same individuals on different days. The total catabolism of the infant is the resultant of two factors: first, the metabolism due to the internal activity incidental to circulation and respiration and the general muscle tonus of the body, that is, maintenance metabolism; second, the metabolism due to the external muscular activity, which may vary from slight movements of the hand or fingers to violent movements incidental to severe crying.

The external muscular movements are recorded with considerable fidelity on the kymograph drum by means of the registering apparatus described, but we have seen that this record does not give a comparative picture of the degree of activity of different infants. Consequently for comparing the maintenance metabolism only periods in which the external muscular activity is eliminated should be used, since in the last analysis, knowledge with regard to internal muscular activity is desired, uncomplicated by the increased metabolism due to external muscular activity. We believe that our evidence justifies us in asserting that we have two admirable indices for securing these ideal conditions of muscular repose for comparison, first, the graphic records obtained with the kymograph, and second, the pulse-rate. For comparing the metabolism of different infants, therefore, only those periods with records of complete muscular repose and with a minimum pulse-rate can legitimately be employed.

5. Relationship between Pulse-Rate and Metabolism

From the general pictures of the kymograph curves and the pulse-curves, one may infer that the pulse-rate follows closely the muscular

activity. Furthermore, since it has been shown that the relationship between the metabolism and the kymograph curves is comparatively constant, it is reasonable to expect that the pulse-rate will follow the metabolism. That this latter relationship is usually more nearly constant than the relationship between the metabolism and the record of the muscular activity is clearly indicated in a number of observations in which the kymograph record showed a complete absence of extraneous muscular activity while the pulse-records showed fluctuations.

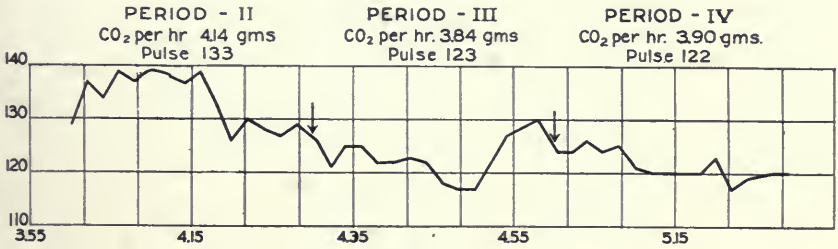

Fig. 8a

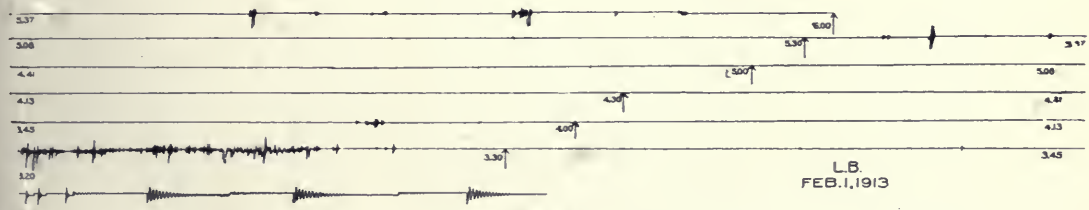

Fig. 8b

Fig. 8.—Pulse-rate and kymograph curves for L. B., Feb. 1, 1913.

An excellent illustration of this may be seen in the pulse and kymograph curves which were obtained in the observation of Feb. 1, 1913, with the infant L. B. (Fig. 8). Thus between 4 p. m. and 5:30 p. m., one may assume that the activity was essentially of Grade I, that is, minimum. Nevertheless the pulse-rate is considerably higher in the period between 4 p. m. and 4:30 p. m. than in the two following periods, as is shown by the pulse-curve and the figures for the pulse-rate per minute. As a matter of fact, the total metabolism is likewise higher in the first period as is evidenced by the carbon dioxid output per hour which is given on the pulse-curve. In this curve, therefore, which excludes the extraneous activity, we find the pulse-rate following very closely the total metabolism. While the kymograph

curve did not indicate muscular activity, nevertheless the pulse-rate gave evidence of an increased internal activity.

6. Basal Metabolism of Infants Studied

In the beginning of this research of infant metabolism, one of the fundamental questions which presented itself to us with special force was as to what may be considered the normal basal metabolism of infants. Consequently we made it our aim to study as many infants as possible and to secure a sufficient number of periods of complete repose on a sufficient number of days to establish beyond reasonable doubt the basal metabolism of each infant. The infants secured for these observations varied sufficiently in age, weight, height, and sex to permit a comparative study of the results as to the constancy or lack of constancy in the metabolism.

A. Selection of Data Used for Comparison

From the data obtained a table has been compiled which gives the average results of the periods with each infant in which the metabolism was at a minimum (Table 15). The selection of the periods was based on the records of the pulse-rate and the muscular activity, only such periods being used as showed a normally low pulse-rate and practically no muscular activity, that is, those characterized as I or II. Of the eighty-three infants studied thus far in this research, but sixty were used in the comparison of the metabolism of different infants and but 360 periods out of the 1,250 periods were available for the comparison. The data are arranged according to the increasing weights of the infants. Since with one infant, J. V., the studies continued over a period of several months, the average minimum metabolism is given for periods secured at an early age, and again for periods obtained several months later. Various bases of comparison may be used, but in this table the infants have been compared on the basis of the energy transformation in twenty-four hours.

In employing the data in Table 15 for the discussion of the fundamental questions considered, it is necessary to emphasize the fact that the amount of material and the method of its selection justify its use for a basis of comparison.

The number of infants (sixty in all) permits extended comparison and discussion.

Furthermore, the data are sufficiently extensive for each subject, as an examination of the table will show that in all but two instances, at least two periods are used for securing the average value for each infant; in other words, the values were determined in duplicate. In many cases the number of periods for comparison greatly exceeded this; for example, in one instance twenty-two periods were available for averaging.

Obviously no infant lives on a minimum metabolic plane throughout the entire twenty-four hours; indeed, but a small proportion of the total number of the experimental periods could be utilized for this important comparative study. Nevertheless, since in but two instances was it necessary for us to rely on the computation of the minimum metabolism of the infant from one experimental period, and in only one other instance were our data limited to those secured in two periods on one day, we believe that we have obtained a reasonably accurate estimate of the minimum metabolism of each infant which justifies critical study and comparison.

B. Minimum Influence of Food

In discussing our results, the criticism can be raised that one of the factors outlined in our definition of basal minimum metabolism was not so strictly observed in this study as could be desired in that the infants were rarely in the postabsorptive state, since the observations were for the most part made from one to one and a half hours after the ingestion of food.

It has clearly been shown in experiments on men and animals that the ingestion of a mixed diet results in an increased metabolism. When isolated nutrients are ingested, the greatest increase has been observed with protein. With fat there is relatively but little, if any, increase. With carbohydrates, while investigators differ as to the quantitative relationships, it has been observed with men in this laboratory that cane-sugar and levulose may stimulate the metabolism to a degree comparable with that resulting from the ingestion of an equivalent weight of protein. On the other hand, lactose—the chief carbohydrate in the diet of infants—has a minimum influence on the metabolism.

This criticism of our experiments has, therefore, considerable theoretical importance, but practically we must consider the fact that the diet of the infant is of such a character as to produce a minimum amount of increase in the metabolism. With infants a large propor-

TABLE 15.—MINIMUM—

Subject	Sex*	Body-Weight Without Clothing kg.	Height cm.	Age	Days Included in Average	Periods Averaged	Carbon Dioxid per Sq. Meter (Meeh) per Hr.†
I. V.	♀	1.94	47	3½ months	2	3	12.1
E. H. S....	♂	2.96	51	3½ months	5	8	11.6
A. C.	♀	2.99	..	1½ months	3	8	9.7
E. S.	♀	2.99	..	5 months	3	6	14.0
A. D.	♀	3.16	56	4½ months	5	15	13.0
K. R.	♂	3.17	56	4 months	2	4	12.1
A. L.	♀	3.18	53	4 months	2	2	12.4
L. O.	♂	3.18	..	6 months	7	12	15.3
J. B.	♂	3.23	..	5 months	2	4	13.3
G. S.	♂	3.30	..	2½ months	3	5	12.1
R. D.	♀	3.31	47	3 days	4	5	7.1
J. V.	♀	3.38	53	8½ months	3	3	16.7
H. C.	♀	3.42	55	5 months	2	4	15.2
O. C.	♂	3.45	51	2 days	3	6	7.8
J. O.	♀	3.53	57	4½ months	2	6	13.8
T. K.	♂	3.60	53	19 hours	2	3	7.5
I. N.	♂	3.65	52	3 days	3	5	7.5
F. M.	♂	3.65	..	4½ months	3	5	15.4
E. C.	♂	3.70	57	2½ months	3	5	13.9
E. P.	♀	3.71	51	12½ days	2	4	7.9
G. M.	♀	3.73	58	6 months	4	8	15.2
I. R.	♂	3.89	52	6½ days	2	2	10.3
M. D.	♂	3.99	..	17 days	2	4	9.2
U. H.	♂	4.00	53	15 hours	2	2	7.4
L. B.	♀	4.04	..	4 months	3	8	13.2
R. C.	♀	4.13	51	37½ hours	2	4	7.9
E. L.	♂	4.15	59	4 months	1	2	14.0
W. P.	♂	4.31	..	5 months	2	6	14.6
F. R.	♂	4.40	52	6 days	6	8	8.6
J. S.	♂	4.41	63	5½ months	5	7	15.6
E. R.	♂	4.49	55	3 months	3	5	12.0
T. C.	♂	4.72	66	8½ months	5	10	15.5
C. N.	♀	4.74	63	4½ months	2	6	13.6
F. D.	♂	4.80	59	2½ months	1	2	12.4
F. B.	♂	4.87	60	5½ months	4	13	15.7
B. D.	♀	4.90	58	2 months	2	4	10.5
R. E.	♂	5.04	60	4½ months	3	7	12.2
L. L.	♂	5.13	57	2½ months	10	13	10.6
D. M.	♂	5.18	66	11 months	2	2	15.0
D. Q.	♂	5.28	62	4½ months	2	4	11.9
E. N.	♀	5.40	66	6 months	7	22	13.9
J. P.	♂	5.45	63	7 months	4	7	15.3

* In this column ♀ means female and ♂ male. † In accordance with the usage of European writers, we give these values, although we believe they are peculiarly liable to misunderstanding and hence their use is unfortunate. ‡ See page 114.

—METABOLISM OF INFANTS

Oxygen per Sq. Meter (Meeh) per Hour gm.	Heat Produced					Average Rectal Temperature During Respiration Periods		Pulse-Rate
	Per 24 Hrs. cals.	Per Kilogram per 24 cals.	Per Square Meter per Twenty-Four Hours			°C	°F	
			Howland‡ cals.	Lissauer $\frac{3}{2}$ $10.3\sqrt{w}$ cals.	Meeh $\frac{3}{2}$ $11.9\sqrt{w}$ cals.			
10.8	164	85	984	1,032	882	....	....	129
9.6	194	65	891	906	783	36.8	98.2	109
8.0	163	55	756	759	660	37.2	98.9	126
11.0	225	75	1,036	1,048	911	36.5	97.7	107
10.7	229	72	1,010	1,026	895	36.5	97.7	114
10.2	213	67	936	960	829	36.6	97.8	103
10.7	226	71	996	1,020	876	36.8	98.3	107
12.1	260	82	1,154	1,172	1,008	36.9	98.5	106
10.2	223	69	978	996	854	36.1	97.0	95
10.0	216	65	931	946	818	36.9	98.4	119
6.8	146	44	632	641	554	37.1	98.8	110
13.4	297	88	1,264	1,280	1,108	37.3	99.1	126
12.6	280	82	1,184	1,199	1,039	37.3	99.2	113
8.0	168	49	703	714	618	36.9	98.4	129
11.2	255	72	1,051	1,069	925	36.8	98.2	104
7.0	158	45	655	664	575	37.0	98.6	105
8.0	174	48	702	715	616	36.9	98.4	104
13.0	300	83	1,219	1,238	1,064	37.1	98.8	118
11.3	267	72	1,068	1,085	939	37.4	99.4	122
7.5	169	46	672	683	590	36.8	98.3	119
12.7	299	80	1,186	1,205	1,041	36.8	98.2	112
8.6	211	54	810	827	715	36.8	98.3	127
8.2	196	49	738	756	656	37.0	98.6	127
6.8	167	42	630	643	556	37.1	98.7	114
10.6	272	67	1,020	1,041	901	36.9	98.5	124
7.6	186	45	684	701	606	37.1	98.8	108
12.3	306	74	1,128	1,152	995	37.2	99.0	127
11.6	303	70	1,076	1,104	962	36.8	98.2	96
8.0	207	47	732	752	649	37.2	99.0	121
11.9	319	72	1,114	1,152	997	36.9	98.5	111
11.0	283	63	979	1,013	873	37.2	98.9	116
12.9	354	76	1,194	1,230	1,062	37.0	98.6	109
12.0	330	70	1,097	1,136	982	36.9	98.4	116
11.0	303	63	999	1,033	892	37.7	99.8	115
13.1	370	77	1,211	1,257	1,082	37.2	98.9	111
9.8	274	57	898	927	802	37.7	99.8	122
11.3	324	64	1,035	1,070	919	37.1	98.7	114
9.4	269	52	844	878	759	37.3	99.1	119
12.5	369	71	1,152	1,188	1,034	37.3	99.1	119
10.1	305	57	930	972	846	37.3	99.1	101
11.7	356	66	1.069	1,117	962	37.1	98.7	111
13.0	387	70	1,152	1,207	1,039	36.8	98.3	105

TABLE 15.—MINIMUM METABOLISM—

Subject	Sex*	Body-Weight Without Clothing kg.	Height cm.	Age	Days Included in Average	Periods Averaged	Carbon Dioxid per Sq. Meter (Meeh) per Hr.†
M. M.	♀	5.47	62	4½ months	3	7	10.6
J. M.	♂	5.63	62	8 months	2	6	18.2
M. A.	♂	5.67	68	9 months	4	9	12.9
F. K.	♂	5.71	65	7 months	6	8	14.2
B. F.	♀	5.72	64	7 months	2	5	14.9
L. R. B.....	♀	5.99	64	4 months	4	11	11.3
A. S.	♂	6.02	63(?)	3 months	1	1	10.6
M. C.	♀	6.17	63	4 months	3	7	11.9
E. M.	♂	6.19	63	8 months	2	2	18.7
R. A.	♀	6.42	65	7 months	2	3	12.4
N. D.	♀	6.79	67	7 months	3	5	12.3
P. S.	♂	6.80	67	12 months	3	10	14.4
E. F.	♂	7.07	62	3 months	2	2§	10.2
P. W.	♂	7.11	64(?)	7 months	2	5	14.2
R. L.	♂	7.58	71	8½ months	5	8	13.9
E. K.	♂	8.03	73	17 months	1	2	14.2
H. T.	♂	9.33	75(?)	5½ months	1	1	12.4
E. G.	♂	9.37	74	10 months	3	5	11.4
R. S.	♂	9.94	74	9½ months	2	4	13.4

§ The activity estimated for these two periods was II and III, respectively.

tion of the protein ingested—some 60 per cent. or more—may be stored in the body, and Rubner has shown that this storage does not affect the total metabolism. Since the protein ingested by the infant rarely exceeds 15 per cent. of the total energy requirement of the body,[50] it can be seen that we may expect from this nutrient only the minimum influence on the heat-production of infants. Fat has admittedly but a slight influence, while the predominating carbohydrate—milk-sugar or lactose—has likewise only a minimum influence.

On these grounds, therefore, one would conclude that the total nourishment of the infant consists of material which for the most part does not tend to stimulate the metabolism greatly. On the other hand, so keen an observer as Schlossmann[51] states that the effect of the ingestion of food probably persists for some eighteen hours. Prac-

50. Rubner: Sitzber. k. Preuss. Akad. Wissensch, 1911, xx, 440.

51. Schlossmann: Atrophie und respiratorischer Stoffwechsel, Kassowitz Festschrift, Berlin, 1912, p. 318.

—OF INFANTS—(*Continued*)

Oxygen per Sq. Meter (Meeh) per Hour gm.	Heat Produced					Average Rectal Temperature During Respiration Periods		Pulse-Rate
	Per 24 Hrs. cals.	Per Kilogram per 24 cals.	Per Square Meter per Twenty-Four Hours					
			Howland; cals.	Lissauer $10.3\sqrt[3/2]{w}$ cals.	Meeh $11.9\sqrt[3/2]{w}$ cals.	°C	°F	
9.7	285	52	854	891	770	36.8	98.2	96
15.1	467	83	1,368	1,432	1,239	37.2	98.9	112
11.7	365	63	1,037	1,085	939	36.9	98.5	105
12.6	381	67	1,107	1,158	1,003	37.2	98.9	109
12.3	386	68	1,115	1,165	1,016	37.1	98.7	109
10.4	331	55	923	973	844	37.2	99.0	106
9.5	305	51	840	888	774	37.3	99.1	113
10.1	333	54	912	967	837	37.1	98.8	103
16.2	535	87	1,452	1,540	1,334	37.4	99.4	130
10.5	358	56	940	1,002	868	37.4	99.3	118
11.1	381	56	965	1,032	893	37.2	99.0	100
13.3	453	66	1,133	1,219	1,058	36.8	98.2	100
8.7	311	44	756	828	708	37.1	98.8	111
12.2	439	62	1,061	1,147	998	37.1	98.8	120
12.2	455	59	1,038	1,140	991	37.4	99.4	115
12.9	497	62	1,092	1,212	1,044	37.7	99.9	105
9.5	420	45	816	912	797	37.2	98.9	101
11.4	479	51	922	1,046	907	37.2	98.9	106
11.5	531	53	971	1,115	965	37.3	99.1	108

tically all of the investigators in metabolism have concluded that with adults, unless the diet is abnormally rich in protein, the metabolism reaches the basal line twelve hours after the last meal.

In our studies, while it was impracticable to secure the metabolism on all of the infants eighteen hours after the last meal, an effort was made to find out the length of time required to obtain the minimum basal metabolism after feeding milk. To this end some five or six infants were studied 1, 2½, 5, 9, 12, 18 and 21 hours after food. The difficulties in securing ideal periods of rest exactly coincident with definite periods of time after the ingestion of food are sufficiently obvious to need no special comment here; it is only necessary to state that our evidence is admittedly not so complete as we should like. A critical examination of the data shows us, however, that on the whole the influence of milk feeding on the metabolism of infants must be very slight. In certain instances the metabolism during quiet periods immediately after feeding is from 5 to 10 per cent. higher than eighteen to twenty-one hours after, while in others the metabolism twenty-one

hours afterward, even in periods of complete muscular repose, was slightly greater than immediately after feeding. But the general picture derived from these observations indicates that the ingestion of milk played a very slight, if any, rôle in affecting the heat-production of the infants studied.

Recent observations in this laboratory during a thirty-one-day fast showed that as soon as food was completely withheld, the body storage of glycogen was rapidly drawn on and when exhausted, a distinct acidosis appeared. Our experience with diabetics and with normal persons subsisting on a carbohydrate-free diet[52] gives evidence that such an acidosis tends to increase the basal metabolism. Additional light has been thrown on this subject by Schlossmann and Murschhauser,[53] who have shown in a recent publication the influence of the withdrawal of food on the excretion by infants of products of acidosis, particularly acetone, diacetic acid, and beta-oxybutyric acid. Even in the first hours of fasting, definite evidence of the excretion of beta-oxybutyric acid shows the beginning of acidosis. Knowing, as we do, that acidosis strongly tends to increase the metabolism, one sees instantly that a point or a moment when the influence of the previously ingested food ceases and the influence of an oncoming, though slight, acidosis begins is extremely difficult, with our present knowledge, to foretell. It should not be overlooked, however, that Schlossmann and Murschhauser did not find an increased heat-production in these infants showing incipient acidosis although we are inclined to doubt the validity of drawing conclusions regarding so subtle a factor as acidosis from periods with such changes in the degree of repose.

While, therefore, we recognize clearly that the presence of food in the alimentary tract of our infants has distinct theoretical objections, we believe that such influence, if it exists, can play no quantitative rôle in the striking comparisons of the basal metabolism of different infants which are made in the subsequent pages.

7. Comparison of Body-Weight and Metabolism

With the abundance of experimental material in hand, which was obtained with different infants, it is obvious that a large number of comparisons may be made in a variety of ways. Perhaps the simplest

52. Benedict and Joslin: Carnegie Institution of Washington, Publication 176, 1912, p. 134.

53. Schlossmann and Murschhauser: Biochem. Ztschr., 1913, lvi, 396.

method of considering the results is on the basis of the total heat-production. Accordingly a chart has been plotted showing the total heat-production of infants of varying weights, the actual weight at the time of the observation being used (Fig. 9). It is normally to be expected that a large animal gives off more heat than a small animal, and consequently we find that there is a tendency for the heavier infants to have a larger heat-production. Yet this is by no means invariably the case, for in a large number of instances, an infant of

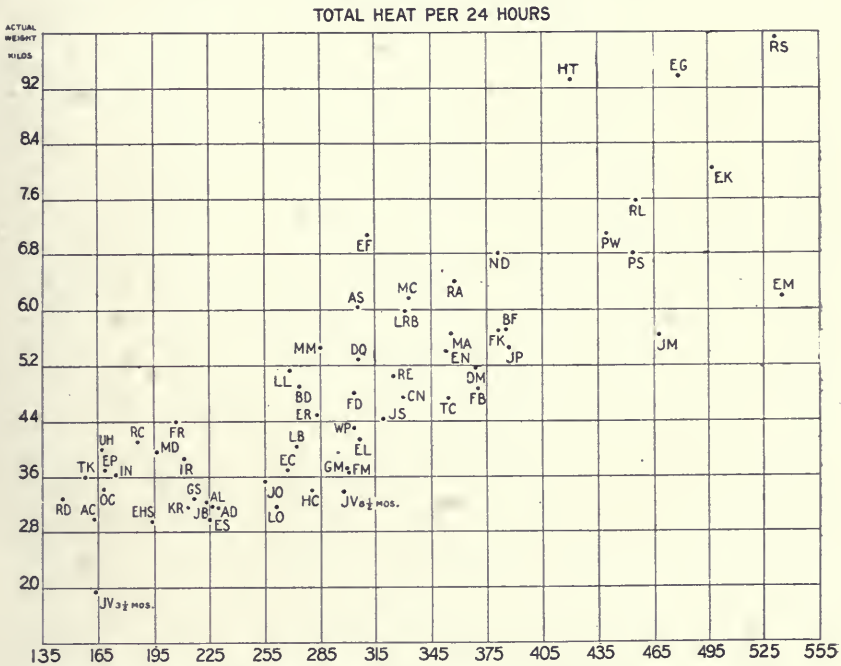

Fig. 9.—Chart showing the actual body-weight of infants and the total heat-production per twenty-four hours.

relatively small weight has a much larger heat-production than those of greater weight. Perhaps the most striking instance is that with the subject E. M., who, with a body-weight of 6.2 kg., has 25 per cent. larger heat-production than has H. T. with a body-weight of 9.3 kg. While it is clear from the chart that in general the large infants show a larger heat-production, yet as there are many exceptions, no definite rule based solely on body-weight can be of great use. Although no

general uniformity is seen at first in this curve, if one examine more particularly the values for the infants indicated as strictly normal in the table showing the clinical status of the infants (Table 13), one can see that there is a distinct tendency to approximate a regular curve. Inasmuch as we are still occupied in overcoming the paucity of results obtained with normal infants, we hardly feel justified in laying sufficient stress on this point to designate specially the normals in our charts.

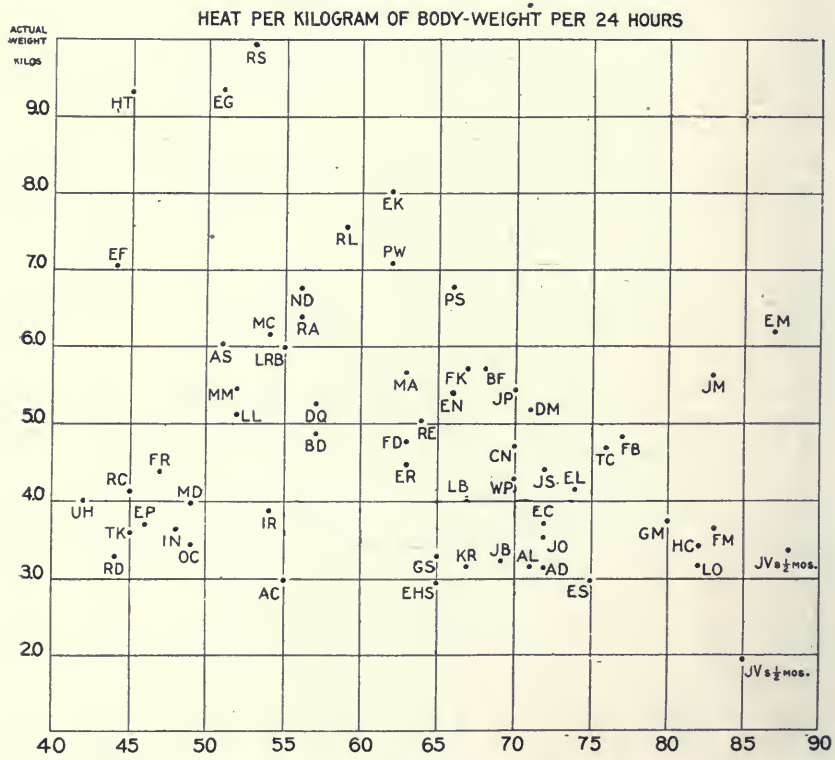

Fig. 10.—Chart showing the actual body-weight of infants and the heat-production per kilogram per twenty-four hours.

It has been the custom of many writers to compare the heat-production of persons of varying sizes by computing the heat-production per kilogram of body-weight, thus tacitly, at least, assuming a constancy in the heat-producing value of each kilogram of body-weight. Although dissenting from this principle, we have, for comparison purposes,

computed the values for our infants on the basis of per kilogram of body-weight and give a part of them in Figure 10. Here there is, as would be expected, a large variation in the heat per kilogram of body-weight and no regularity is apparent. If the "normal" infants are selected, it is found that they, for the most part, lie under 65 calories per kilogram of body-weight, although they may go as low as 42 calories. For an intelligent comparison of the metabolism of different infants, therefore, it is evident that we have to deal with some factor or factors other than body-weight and no definite deduction can be drawn other than the fact that the majority of our "normal" infants have a heat-production per kilogram of body-weight somewhat under 65 calories per twenty-four hours.

8. Comparison of Body-Surface and Metabolism

For many years writers in metabolism have been wont to emphasize the significance of the relationship supposed to exist between the metabolism and the body-surface rather than that between the metabolism and the body-weight. The idea that there is an intimate relationship between body-surface and heat-production was first brought out by Bergmann[54] in 1847. The theory lay dormant for many years, but was finally resuscitated and put forth in a brilliant and highly stimulating manner by Rubner[55] in 1883, together with experimental evidence. Based fundamentally on Newton's law of cooling, it received great attention from practically all workers in physiology. The startling evidence which was brought forward to demonstrate that the heat-production per square meter of body-surface was about 1,000 calories for practically all species of animals lent further support to this hypothesis. In connection with our own researches we naturally expected to find a close relationship between body-surface and total metabolism, particularly in view of the fact that recent observations from foreign laboratories appeared to confirm the validity of Rubner's law. We were, therefore, greatly surprised on preparing our final figures to find this intimate relationship entirely disturbed.

54. Bergmann and Leuckart: Anatomisch-physiol. Uebersicht des Thierreichs, Stuttgart, 1852, p. 272. Bergmann: Wärmeökonomie der Thiere, Göttingen 1848, p. 9.

55. Rubner: Ztschr. f. Biol., 1883, xix, 545.

A. Methods Used for Measurement of Body-Surface

In order to discuss intelligently the relationship between the metabolism and body-surface, a critical examination of the various methods for determining the body-surface is essential. Using as a basis the relationship between the surface of similar solids which is expressed by the cube-root of the square of the weight, efforts have been made by a number of investigators to compute the body-surface of various animals and persons from the body-weight.

Meeh[56] found that he could measure the body-surface of men by using the constant 12.312, which, when multiplied by the cube-root of the square of the body-weight in grams, gave the body-surface in square centimeters. Rubner and Heubner,[57] who first applied this formula to the study of the total metabolism of infants, rightly substituted the value 11.9 which was determined by Meeh on two well-nourished infants under 1 year that he measured.

Recognizing the importance of taking into consideration the length of the body as well as the circumference of breast and abdomen, Miwa and Stoeltzner,[58] using Meeh's measurements, proposed another formula in which the length and circumference as well as weight should appear as factors. This formula has not been generally accepted by research workers.

Actual measurements of the body-surface of cadavers have also been used in an attempt to find some mathematical formula expressing the relationship between body-weight, and body-surface. Lissauer[59] measured twelve cadavers, eleven of which were under 1 year, and found that the constant 10.3 should be used in the Meeh formula instead of those previously proposed. It has been maintained by other writers that since many of Lissauer's measurements were made on thin, poorly nourished, and atrophic infants, they do not give standards for well-nourished infants. Sytscheff[60] measured ten infants under 1 year of age, but computed no ratios. Howland,[43] employing Meeh's and Lissauer's measurements, has recently proposed still another

56. Meeh: Ztschr. f. Biol., 1879, xv, 425.
57. Rubner and Heubner: Ztschr. f. exp. Pathol. u. Therap., 1904-1905, i, 1.
58. Miwa and Stoeltzner: Ztschr. f. Biol., 1898, xxxvi, 314.
59. Lissauer: Jahrb. f. Kinderh., 1902, lviii, 392.
60. Sytscheff: Measure of Volume and Body-Surface of Children According to Their Ages. Dissertation, St. Petersburg, 1902. See also Gundobin, loc. cit., p. 54.

method for computing the body-surface based on a curve represented by the algebraic formula $y = mx + b$.

With these three methods in vogue for computing the body-surface, that is, that of Rubner and Heubner using the Meeh formula with the constant 11.9; that of Lissauer using the constant 10.3 and that of Howland using the algebraic curve, it can be seen that with the great weight laid by all experimenters in infant metabolism on the relationship between body-surface and metabolism, it is incumbent on us to present our results on the three separate bases, although the relative values are unaltered in all three cases. This is done in Table 15.

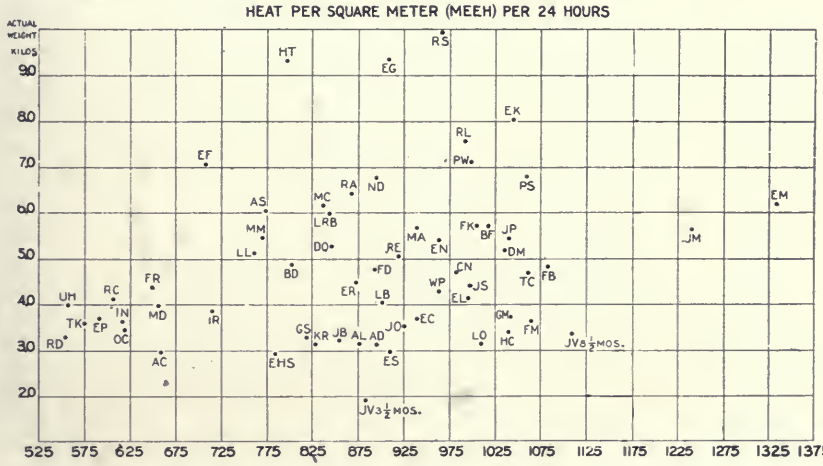

Fig. 11.—Chart showing actual body-weight of infants and heat-production per square meter of body-surface (Meeh formula) per twenty-four hours.

B. Comparison of Actual Body-Weight and Heat-Production per Square Meter of Body-Surface

According to accepted ideas we should expect the heat-production per square meter of body-surface to be approximately constant for all of our infants. That this fact is far from being true is clearly seen in Table 15, but the variations are most strikingly shown if we compare them with the actual body-weight of the infant, as is done in Figure 11. In this chart, which is made up on the Meeh formula, we should expect to find the values grouping themselves in a vertical line. On the contrary, the dispersion of the values is very noticeable, with a tendency, if any, toward a horizontal rather than a vertical

alignment. An actual variation from 554 to 1,334 calories or 140 per cent. is recorded. Omitting the extreme cases, J. M. and E. M., although frankly we see no reason why they should be omitted in this comparison, the range is 554 to 1,108 or 100 per cent. Even "normal" infants range from 554 to 998 calories. Thus the complete absence of correlation between weight and the heat-production per square meter of body-surface is strikingly shown. It is again important at this point to recall the fact that the observations made on these infants were all under constant conditions, namely, complete muscular repose and approximately the same length of time after feeding. It is impossible, therefore, to explain these great discrepancies as due to muscular activity, nor can they in any way be accounted for by the ingestion of food, as our experiments have shown that the food taken by these infants while under observation has no material influence on the metabolism. Although we are deferring its discussion until a later article, it is of interest to point out here the low heat-production per square meter of the numerous new-born infants.

9. Effect on Metabolism of Possible Disturbance in Relationship Between Body-Surface and Body-Weight

It has frequently been the custom when discrepancies in the heat-production per square meter of body-surface are found with infants, and particularly with atrophic infants, to ascribe the variation to a disturbance of the relationship between body-surface and the body-weight from which it is computed. It is essential, therefore, at this point to discuss this possibility more in detail.

The argument frequently raised is that disturbances in the relationship between body-weight and body-surface with underweight infants precludes the use of any of the formulas now regularly used for the computing of body-surface, in that they give too small a value of body-surface for such infants. At the outset we wish to oppose this general thesis on the ground that in the most extensive and remarkably accurate series of measurements on infants with which we are familiar, namely, those of Lissauer, it is especially emphasized that ten out of twelve of the infants were very much under weight. This will be seen by reference to Table 16, which reproduces the weights of eleven of the infants measured by this investigator.[59]

As Lissauer himself points out with regret, S-i was the only infant that could be called normal, although S-r was practically of normal

weight. All of the other infants were noticeably under weight, far more so than our infants as a rule. Yet, in spite of this great deficiency in weight, the relationship between the body-weight and the measured body-surface was represented by the difference between the constant 10.3 used by Lissauer and 11.9, the constant of Meeh. In other words, this large variation in weight produced a maximum discrepancy of not over 15 per cent. in the relationship between the body-surface as actually measured and the body-weight. We believe that Lissauer's for-

TABLE 16.—BODY-WEIGHTS OF INFANTS MEASURED FOR BODY-SURFACE BY LISSAUER

Name	Sex	Age	Body-Weight kg.	Avg. Weight for Age Obtained from Heubner, kg.
M-f	M	3½ months	3.27	6.2
H-r	F	3½ months	1.96	6.2
R-e	F	3¾ months	3.37	6.3
S-e	M	1 month	2.22	4.008
S-r	F	1 month	3.83	4.008
M-r	M	15 months	5.23	11.+
W-t	M	17 days	1.73	3.6
S-t	M	22 days	1.28	3.7
P-z	M	3¼ months	2.50	6.0
H-z	M	7½ months	3.10	8.2
S-i	M	3⅔ months	6.18	6.3

mula, in general, more nearly fits the requirements of observations in clinics, where the larger number of infants are under weight. On the other hand, as we have already pointed out, it is distinctly questionable whether the methods of measurement[61] have even yet been sufficiently

61. As an interesting evidence of our initial belief in the importance and significance of the measurement of body-surface and its relationship to metabolism, we should here state that extensive preparations were made by us for the measurement of the body-surface of a number of infants, and a method was developed for securing shadow photographs of infants in various positions, the areas of the shadows being measured by a planimeter. It was our hope to establish thereby some relationship with the body-surface as measured from the shadow photograph, and by actual measurements of cadavers, and the body-weight and length. It is needless to say that with our present views in regard to the significance of body-surface in the relation to metabolism we have not felt justified in continuing such a series of measurements.

refined or are sufficiently numerous to give a reliable method for the computation of the body-surface from the body-weight.

Although we believe that the lack of consistency exhibited by our infants in the heat-production per square meter of body-surface may not be ascribed to the fact that these infants were distinctly under the average weight, it is of special interest to select a few infants who are of normal average weight and note the relationship between the heat-production and the body-surface. This has been done in Table 17, in which the heat-production per square meter has been calculated for eight of our normal infants of average weight.

TABLE 17.—HEAT-PRODUCTION PER SQUARE METER OF BODY-SURFACE (MEEH FORMULA) FOR NORMAL INFANTS

Subject	Body-Weight Without Clothing kg.	Height cm.	Age	Experimental Days	Periods	Heat per Square Meter of Body-Surface (Meeh) cals.
M. D.	3.99	..	17 days	2	4	656
L. L.....	5.13	57	2½ mos.	10	13	759
B. D.....	4.90	58	2 mos.	3	4	802
M. C.....	6.17	63	4 mos.	2	7	837
L. R. B....	5.99	64	4 mos.	4	11	844
E. G.....	9.37	74	10 mos.	3	5	907
R. L.....	7.58	71	8½ mos.	5	8	991
P. W.....	7.11	64 ?	7 mos.	2	5	998

10. Influence of Variations in the Composition of the Body on Total Heat-Production

Since a gross disturbance in the relationship between the body-weight and the body-surface as computed from the body-weight is highly improbable whether the infant is atrophic or well-nourished, it is important to find out, if possible, if any relationship exists between the general composition of the body and the total heat-production. Our data are sufficiently extended to permit a somewhat incomplete discussion of this important phase of the comparisons.

Heretofore, all workers in metabolism have considered only the relationship between body-weight and metabolism, or body-surface and metabolism. Since the body-surface is assumed to have a direct

relationship to the body-weight, it can be seen that body-weight is the only fundamental factor which has thus far been seriously considered by investigators in comparing the metabolism of different infants.

TABLE 18.—COMPARISON OF HEAT-PRODUCTION OF INFANTS OF LIKE BODY-WEIGHT AND HEIGHT, BUT OF DIFFERENT AGES

Subject	Sex	Body-Weight kg.	Height cm.	Age mos.	Heat Produced		
					Per 24 Hours cals.	Per Kilogram per 24 Hrs., cals.	Per Sq. Meter (Meeh) per 24 Hrs., cals.
A. L.....	F.	3.18	53	4	226	71	876
J. V.....	F.	3.38	53	8½	297	88	1,108
E. N.....	F.	5.40	66	6	353	66	962
D. M.....	M.	5.18	66	11	369	71	1,034
M. M.....	F.	5.47	62	4½	285	52	770
J. M.....	M.	5.63	62	8	467	83	1,239
D. Q.....	M.	5.28	62	4½	305	57	846
J. P.....	M.	5.45	63	7	387	70	1,039
M. M.....	F.	5.47	62	4½	285	52	770
J. P.....	M.	5.45	63	7	387	70	1,039
D. Q.....	M.	5.28	62	4½	305	57	846
J. M.....	M.	5.63	62	8	467	83	1,239
L. R. B...	F.	5.99	64	4	331	55	844
F. K.....	M.	5.71	65	7	381	67	1,003
H. T.....	M.	9.33	75 ?	5½	420	45	797
E. G.....	M.	9.37	74	10	479	51	907

It is obvious that when two infants are of the same weight, the shorter one will have the larger proportion of fat. Furthermore, with two infants of the same length but of different weights, the heavier infant will have the larger proportion of fat. It can be seen, therefore, that an atrophic infant, weighing 4 kg. and 65 cm. long, when compared to a well-nourished infant of the same weight and length, would have a smaller proportion of fat. Moreover, an atrophic infant, to have the same weight and length as a normal infant, must obviously

be older, and we here find a new factor entering into the comparison of infants; as yet the element of age has received scant attention. An inspection of Table 15 will show that in a number of instances infants with approximately the same body-weight and the same height differ greatly in age. Unfortunately our data are not so extensive as to enable us to compare infants with absolutely the same body-weight and height, but a number of comparisons are justifiable and these have been included in Table 18.

In this table eight series of comparisons are made of the total heat produced, the heat-production per kilogram of body-eight, and the heat-production per square meter of body-surface for infants with the same body-weight and height but of different ages. The difficulties incidental to measuring exactly the length of infants make these measurements slightly problematical and there may be a variation of plus or minus 1 cm. We have, therefore, compared infants whose lengths do not vary more than 1 cm. The variations in weight are all within a few tenths of a kilogram.

We note instantly several striking points in the data as presented. In each comparison the values for the younger infant are given first, and it will be seen that the older infant has invariably the larger total heat-production. The greatest difference is 182 calories in the comparison of M. M. with J. M., the lowest difference being that of 16 calories between E. N. and D. M. Aside from this latter comparison, the increase in the heat-production for the older infants is very considerable. The heat-production per kilogram of body-weight and per square meter of body-surface also shows this increase in the same general proportion since the body-weights of the infants compared are essentially the same in all cases.

In the two series of comparisons in which the youngest infant is approximately 6 months old, namely, those comparing E. N. with D. M. and H. T. with E. G., the increase in the heat-production for the older infant is not so great. In the latter comparison, E. G. was of normal weight while H. T. was over weight so that the excessive amount of fat actually lowered the total heat-production of the younger infant H. T. It is therefore clear that with the older infants, which were in most instances distinctly under weight, there was a deficiency in the fat with an accompanying increase in the proportion of active protoplasmic tissue. While this method of comparing the metabolism of infants on the basis of weight, height and age gives a

clue to the probable preponderance of fat or active protoplasmic tissue, it is obvious that no quantitative relationship can be established on this basis.

The striking comparison between M. M. and J. M. is particularly worthy of consideration, inasmuch as the value for M. M. is derived from observations on three days, and a total of seven satisfactory periods were available for averaging, while with J. M. the data were secured on two days with six periods for comparison. Here, with a difference of 3½ months in the age, there was obviously a much greater proportion of active protoplasmic tissue with the older infant, J. M.

That the active protoplasmic tissue determined to a very considerable extent the total catabolism, not only with J. M., but with all of the older undernourished infants, is highly probable and we find ourselves thoroughly convinced that the metabolism is determined not by the body-surface but by the active mass of protoplasmic tissue. With normal infants of varying weights, it is quite probable that the active mass of protoplasmic tissue varies directly with the age. Since it has been shown that not only body-surface but also more recently the blood-volume, the size of the aorta, and the size of the trachea with several species of mammals bear a direct relationship to the cube root of the square of the body-weight,[62] it is not surprising that most experimenters have observed that with adults the metabolism is roughly proportional to the body-surface. If the blood-volume and the area of the trachea and the aorta are proportional to the cube root of the square of the body-weight, it is reasonable to suppose that the active mass of protoplasmic tissue may develop normally on this ratio. When there are marked variations from the average, as with excessive or with deficient adipose tissue, this relationship cannot be expected to hold.

If, therefore, it is maintained that the total metabolism is proportional to the body-surface, it should be stated that this is not due to the fact that there is a loss of heat from the body-surface and that Newton's law of cooling determines the intensity of the metabolism, but that with normal persons the body-surface, blood-volume, the area of the trachea and aorta, and probably the active mass of protoplasmic tissue, are all in simple mathematical relation to the

62. Dreyer and Ray: Phil. Tr., 1909-1910, cci, ser. B, 133; Dreyer, Ray and Walker: Proc. Roy. Soc., London, 1912-1913, lxxxvi, Ser. B, 39 and 56.

body-weight. Thus the apparent relationship which has previously been observed between the heat-output and the body-surface with normal or nearly normal persons has an explanation in that with such persons a simple relation exists between the body-surface, blood-volume, body-weight, and the mass of active protoplasmic tissue.

In our series of observations we have attempted to eliminate completely all muscular activity, to make the experiments under approximately the same conditions as to nutriment, to select such a diet as was least stimulating to the catabolism and to have our subject for the most part in deep sleep, thus eliminating psychic disturbances. With these conditions we hoped to obtain the fundamental minimum metabolism, on which we might base our discussion.

The basal metabolism as we have outlined above, cannot in any wise be considered a direct function of the body-weight and the body-surface and particularly has no relationship with body-surface on the basis of the law of cooling bodies.

We believe that our evidence points strongly and conclusively to the fact that the active mass of protoplasmic tissue determines the fundamental metabolism. The absence as yet of a direct mathematical measure of the proportion of active protoplasmic tissue does not, we believe, in any wise affect the convincing nature of our evidence.

DISCUSSION ON PAPERS OF DR. GITTINGS AND DRS. BENEDICT AND TALBOT

DR. DUNN: From clinical observations at the Infants' Hospital, I long ago became convinced of the truth of Dr. Talbot's conclusions concerning the theory of the minimum caloric requirement, although I have never been able to see the reason proved as definitely as this. For some time we have been tabulating, at the Infants' Hospital, the caloric value of the diet taken by the babies in the wards, and the result of that tabulation has been somewhat different from the results that Dr. Gittings has reported. He reported that the extremes were not so great as to impair the value of the conclusions based on averaging the different figures. In our results just the reverse has been true. Whether we look at the cases from the point of view of the minimum calories per kilogram of weight on which the baby could gain, or from that of the actual calories taken irrespective of gain or loss, the extremes have been so great that it seems to me that no conclusions based on any system of averaging can be of any value. In fact, I have always felt that the work from which we derive our views as to the caloric requirements of the baby has been based on a false method. That false method is the method of taking a number of approximately normal babies and averaging the results. That was the method by which the work on minimum caloric requirements was done. It is quite easy to conceive that if in such a series there were very wide extremes, the result of the average which would be given as a minimum caloric requirement would be entirely false, and might be, in an extreme instance, wrong for each baby in the series.

At the Infants' Hospital we have tabulated the calories per kilogram of body-weight of all the babies which were gaining weight, and especially those babies which, after a period of failure to gain, begin to gain weight steadily—estimating for each the calories per kilogram of body-weight which they were taking. In these cases the extremes are so enormous that it has seemed as if no possible conclusions could be drawn. They have apparently borne no relation to the clinical aspect of the case. A certain number of babies gained on a very low caloric value; a number of babies who consistently lost on food of 100 or more calories per kilogram of body-weight, began to gain consistently and rapidly when their calories were put down to 30 to 40 in twenty-four hours; and a number of others failed to gain until the caloric value of the food was pushed to sometimes double that of the so-called minimum caloric requirement. In other words, the whole question is so complicated with nutritional variations and variations in digestive power in various types of cases that it seems to me that the conclusions drawn as to the minimum caloric requirements are hardly of value, even as a guide, in the average infant feeding case.

DR. CHAPIN: I am very glad to hear these remarks of Dr. Dunn; because I feared I would be the only one, as I think I was the first to protest against the stress placed on caloric value in infant feeding. There has been, in my opinion, an overestimate of the value of this method; after all, heat is an excretion. The principal thing that we have to consider in an infant food is its digestibility, its nutritive value and its power of assimilation, and not its heat-producing value; and I feel so strongly on that, that in 1910 I wrote an article on "The Limitations of the Caloric Method of Infant Feeding," and I would like to repeat one paragraph from that article.

"In selecting food a number of factors must be considered. First of all, is the food digestible by the *particular individual?* Food for a cow is entirely unsuitable for a dog, and the food of an adult will kill an infant. Yet such foods are nutritious and digestible in their proper spheres.

"Fats, sugars, starches, meat, and other forms of protein will all produce heat, and all are digestible. If the heat or caloric value was all infants or adults needed food for, it would be immaterial what article of diet was employed. But foods that are of equal heat-producing value, and which are also digestible, are not interchangeable for different individuals, and growth and tissue repair do not depend upon the storage of heat, but upon the assimilation of protein. In infant feeding, particularly, where growth or the storage of protein is the chief phenomenon of nutrition, the main point to be considered is not, will the food supply heat, but, is it capable of causing growth?"

DR. VEEDER: Dr. Talbot's paper upsets the ideas of physiology that have been held for years, that is, Dr. Talbot is practically the first to take an opposite point of view of the relation of the body surface to caloric requirements. One or two questions I would like to ask him. One is, what method did he use to calculate the body-surface? Secondly, if according to his idea, the caloric requirement is dependent on protoplasmic activity, is not that just saying in another way that he is comparing babies doing different amounts of work?

DR. JACOBI: What would be the relation of a simply small baby, and an atrophic baby, atrophic in consequence of diseased conditions? The babies may be of the same weight; one baby is simply of minor weight, being well; the other atrophied in consequence of sickness?

DR. FREEMAN: Dr. Talbot's work is perhaps the first scientific work on caloric feeding that corresponds closely to our clinical experience. The marked

variation in the amount of food required by different infants, the fact that some fat babies require little food, and that many poor, marasmic babies require a great deal, all these facts are what we are constantly seeing in practice.

DR. KERLEY: Our inability to establish a calorimetric standard is best demonstrated in the runabout child. An active nervous child will take and require two or three times the amount of energy food that a quiet indolent child of the same age and weight will. This I have demonstrated in private work time and again.

DR. GITTINGS: I did not draw any definite conclusions in my paper. I wished to see what was the deficiency in the given diets. Generalizations from averages are dangerous and I have not attempted to make any. There were few extremes in this series and I presented it chiefly for discussion.

DR. TALBOT: One of the arguments used to explain such findings as those just reported is that atrophic babies have more heat because they have more body surface. Their skin is said to be in wrinkles, which would naturally make more body surface. Lissauer complained, in his original article, that out of thirteen babies he had only one normal baby. All the rest were atrophic, so that Lissauer's formulas were based principally on atrophic babies.

Meeh's formula was based on the measurement of two normal babies. When we started our work we devised a method of obtaining body surface by shadow photographs, or measuring them by perimeter. As the work progressed we came to the conclusion that such measurements would be of no value as there was apparently no connection between the heat loss and the body surface.

THE FEEDING OF MALNOURISHED INFANTS ON HIGH PROTEIN AND CARBOHYDRATES AND LOW FAT VALUES

D. J. MILTON MILLER, M.D.
Associate Physician to The Children's Hospital of Philadelphia.
ATLANTIC CITY, N. J.

This paper lays no claim to be other than a clinical one; no metabolic studies or chemical researches are offered, or theories presented, as to the causes of chronic malnutrition in infants. Nor is there anything original in the method described as fairly successful in the writer's hands in the management of these all too common cases. The principles involved have been repeatedly employed, with more or less success, by many other observers, especially since the more recent conceptions of the rôle of the different milk constituents in the infantile digestive economy has found a place in the minds of physicians.

The feeding of marasmic and malnourished infants has ever been a stumbling block and reproach to pediatrists. The existence of these babies has contributed largely to the popularity of the proprietary foods; has strengthened the belief, still entertained by many, that there is and can be no successful or scientific artificial feeding—that it is all haphazard; and has made it so difficult, as many of us have found, to convince hospital managers that baby wards are worth the cost of their management, so disheartening are the results frequently obtained therein.

The most successful method of nourishing infants in a state of malnutrition has been in the past, and now is, human breast milk; but it has taken the profession at large, and even pediatrists themselves, a long time to grasp the selfevident proposition: Entirely too much time continues to be wasted in finding a suitable food before wetnursing is resorted to. Proprietary foods have always occupied a large place in the feeding of these babies, and it cannot be gainsaid by an unbiased observer that many apparently hopeless cases react surprisingly well to some of these preparations, particularly the condensed, dried and malted varieties. Every one of us must have had this experience.

Curiously enough, malnourished babies are the ones in whom undiluted cow's milk has been most often successful, possibly because of its

relatively low fat and sugar and high protein content. In recent times, buttermilk, albuminmilk and Keller's malt soup have been extensively and often successfully employed, but the most frequent method of dealing with these infants, particularly since the advent of percentage feeding, has been the exhibition of low fats and proteins and relatively high sugar values. Albuminmilk, just referred to, appears also to be of great value, but it is only applicable for brief periods, as a preparation for a more rational dietary.

When one scrutinizes the foods and methods of feeding which have been more or less successful in malnutrition and marasmus, several points of importance will be noted, namely: The low fat, and, frequently, high carbohydrate content of these preparations, and the attenuated or altered state of the casein, even though the latter may not be present in high amount. Thus are explained the good results sometimes seen after condensed milk, albuminmilk, Keller's preparation and some of the proprietary foods, and thus is the way indicated for the employment of other and more generally applicable methods of feeding.

Whatever may be the essential condition that prevents malnourished babies from utilizing the food elements presented to them, they, in most cases, *at least at the time when they usually come under observation,* give marked evidence of an intolerance for cow's milk fat; not infrequently they are taking formulae in which the fat is in amount suitable to their age and weight; quite often it is abnormally high, and yet the infant fails to utilize it and remains undersized and puny. Whether this failure is due to an inherent or acquired inability (through faulty feeding), or to the deleterious influence of the salts and lactose, and is, therefore, a secondary phenomenon, will not be discussed here. The fact remains that, while with normal infants there is usually little trouble in digesting cow's milk fat, with atrophic and malnourished babies it is frequently quite different.

Notwithstanding this, we have found that, after a period of low fat and high protein feeding, these cases can, in many, indeed, in the majority of instances, be rendered immune to the injurious influence of cow's milk fat by making minute increases at short intervals, until an amount sufficient for their nutritive needs is given and well borne. This point is important, as no child can be fed with impunity for pro-

1. New York Med. Jour., 1913, xlvii, 269.

longed periods on a fat-free diet. Dr. H. D. Chapin's[1] observations on the function of fats in nutrition and on metabolic water are most instructive in this particular.

When the fat is reduced to a minimum the protein may be increased relatively, and, in many cases, absolutely, to meet the nutritive and calorific requirements of the infant, as mere increase in the carbohydrates is not sufficient for this purpose. There seems to be no difficulty in the utilization of this element, especially when separated from the fat. Finkelstein's views on this matter are now so generally accepted that further discussion seems unnecessary. I, myself, am convinced of them. No protein injury (fever, etc.) is ever observed in the amounts advisable in ordinary cases of malnutrition; besides, in my cases the percentages of protein were never excessive, and were further protected by the whey in the milk, which Holt and Levene believe tends to prevent the so-called protein fever.

As is well known, proper gain in weight must, as a rule, be secured by the addition of carbohydrates, when the protein is given in safe and rational amounts. In the cases with which this paper deals maltose was the preferred sugar, but both lactose and saccharose were found useful, especially when dextri-maltose proved constipating, as is sometimes its tendency.

At no time did apparent harm result from the use of sugars, other than maltose, in undernourished infants; indeed, it may be stated here that we very much doubt the activity of the sugars in this respect. Our experience is entirely clinical, but beyond fermented and acid stools, *readily corrected, usually,* we have not been impressed by the injurious effect of the sugars in infant feeding.

It was further discovered that in many of our cases more rapid increase of weight could be obtained, the nutrition of the infant better conserved and the stools take on a more natural appearance, if a small quantity of wheat flour (½ ounce to the quart of water, 1.5 per cent.) was added to the dietary. Usually this was dextrinized. The starch in these quantities caused no disturbance. "Starch injuries" were not noted. Occasionally more than one sugar was used: lactose and maltose or maltose and saccharose, as some infants seemed to thrive better when so fed.

Although the term "marasmic" is employed in the title of this paper, it should be stated that the actual cases of which it treats do not belong strictly to this category; not one exhibited the extreme

wasting of marasmus or atrophy. The difference, however, was one of degree rather than of kind; all were much underweight—from 6 to 10 pounds—all exhibited the digestive disturbances, the nervousness, sleeplessness, the tendency to "colds" and adenoid growths so common in this class of infants.

METHOD OF FEEDING

Usually the infants were started upon a fat percentage of 0.50 per cent., with protein at two and one-half to three times, and carbohydrates ten to twelve times greater, *i.e.,* fat, 0.50 per cent.; carbohydrates, 5 to 6 per cent.; protein, 1.30 to 1.50 per cent. The fat and protein were then increased rapidly, every fifth, sixth or seventh day; the fat in tenths of 1 per cent., the protein in fifths or thirds of 1 per cent., while the carbohydrates were at once increased, if no gain in weight was noted, to 7.5 or 8 per cent. Not very marked gains were secured until the latter elements had reached these proportions. The protein was also rapidly increased, depending upon the weight curve, but rarely exceeded 3 per cent. This, however, bore a close relation to the length of time the infant was under observation, and was, until fat tolerance was more or less established, always considerably in excess of the former.

So soon as the baby had begun to gain well and the stools become normal, or nearly so (not before), an attempt was always made to increase the fat in the manner described (*i.e.,* fractions of per cent. every fifth, sixth or seventh day) until two or more per cent. was reached; and this was generally found feasible, provided the conditions stated above were observed, *i.e.,* after the stools were normal and the infant beginning to gain. The quantities of food given were small at first, but it was soon found necessary to increase them to amounts somewhat above that required by healthy infants of the same age, and very *considerably larger than that demanded by their weight.*

The calorific value of the food taken averaged from 60 to 65 per pound. A very few took 70 to 75, while others made good gains on from 45 to 50 per pound, of body weight. Here, as in normal babies, great differences were observed in this respect, demonstrating anew the uncertainty of this method of feeding as a routine procedure.

MODE OF PREPARING THE FOOD

All the patients were seen in private practice, and hence the preparation of the food was made as simple and as inexpensive as

possible: Skimmed milk, containing from 1 to 1.75 per cent. of fat was the basis. When a flour gruel was the diluent, it was dextrinized. To this dextrimaltose, milk or cane sugar was added, as the individual case seemed to require. Sometimes one of the semi-solid malt extracts was employed instead of dextrimaltose (one ounce to the quart). Quite frequently the mixed skimmed milk, malt extract and flour were boiled for a moment, after the manner of Keller.

The results of this method of feeding were usually apparent at once: The stools, which had been very dry, crumbly and clay or light colored, or otherwise abnormal, became light brown and putty-like, gas and pain disappeared, and the infant, as soon as the protein and carbohydrates were sufficiently high, became more cheerful and less nervous and sleepless.

That a food so deficient in fat could be continued any length of time without detriment to the infant goes without saying. Not one of my cases was long enough without fat, or on low fat, to test this point. An attempt was early made to get them upon a more rational dietary, especially upon a fair proportion of fat; although this element in some of the cases followed had to be always kept below the protein of the formula.

The following are brief case reports of infants fed in the manner above described:

CASE 1. *History.*—C. C.: 7 months; birth weight, 7½ pounds; present weight, 7 pounds; average weight for age, 17½ pounds; never nursed; fed upon proprietary foods and top milk mixtures; now gets equal parts milk and water, with a proprietary dextri-maltose, caloric value of which is 65 per pound; given skimmed milk, malt extract and flour brought to a boil; five to six loose, yellow stools daily. No regurgitation.

DATE:	Feb. 5	Feb. 17	Feb. 25	Mar. 13	Mar. 27	Apr. 21	May 1	May 20
Percentages: F.	.50	.50	1.00	1.25	1.50	1.75	2.00	2.00
P.	1.30	1.50	1.90	2.20	2.40	2.75	3.00	3.25
C.	7.	7.	7.	7.	7.	7.	7.	7.
			WEIGHT					
Pounds	7	7½	7¾	8⅛	8 15/16	9 7/16	10½	11
			QUANTITY					
Ounces	40	42	42	46	48	50	50	50

Age at completion of treatment, 9 months; duration of treatment, 3⅓ months; gain, 4 pounds.

CASE 2. *History.*—M. K.: 4 months; birth weight, 6¾ pounds; present weight, 8 pounds; average for age, 14 pounds; nursed one month; now takes: fat, 3.25 per cent.; proteids, 1.30 per cent.; carbohydrates, 5 per cent.; hard, dry, light-colored stools; given dextrinized gruel, skimmed milk and dextrimaltose.

DATE:	Nov. 13	Nov. 18	Nov. 24	Dec. 1	Dec. 8	Dec. 15	Dec. 21	Dec. 25
Percentages: F.	.60	.70	.70	.80	.90	1.00	1.00	1.20
P.	1.30	1.50	1.60	1.75	1.85	2.10	2.25	2.40
C.	6.50	7.50	7.50	7.75	8.00	8.00	8.00	8.00
			WEIGHT					
Pounds	8	$7\frac{15}{16}$	$8\frac{1}{8}$	$8\frac{1}{2}$	$8\frac{15}{16}$	$9\frac{1}{4}$	$9\frac{3}{4}$	$10\frac{1}{4}$
			QUANTITY					
Ounces	32	32	32	36	36	42	42	42
Calories:								
Per pound	43	53						

DATE:	Jan. 1	Jan. 6	Jan. 11	Jan. 16	Jan. 23	Feb. 7	Feb. 14	Feb. 21
Percentages: F.	1.25	1.35	1.45	1.55	1.75	2.00	2.25	2.25
P.	2.50	2.60	2.75	3.00	3.00	3.25	3.25	3.25
C.	7.75	7.50	7.50	7.50	7.50	7.50	7.00	7.00
			WEIGHT					
Pounds	$10\frac{3}{4}$	$11\frac{1}{4}$	$11\frac{3}{4}$	$12\frac{1}{4}$	$12\frac{3}{4}$	$12\frac{3}{4}$	$13\frac{1}{8}$	$13\frac{3}{4}$
			QUANTITY					
Ounces	42	42	45	45	45	45	45	45
Calories:								
Per pound.....	60				65		60	

Age at completion of treatment, 7 months; duration of treatment, $3\frac{1}{4}$ months; gain, $5\frac{3}{4}$ pounds.

CASE 3. *History.*—M. H.: 3 months; birth weight, $6\frac{3}{4}$ pounds; present weight, 8 pounds; average for age, $13\frac{1}{4}$ pounds; nursed a short time; fed upon cream, whole milk and barley; now gets: fat, 3 per cent.; protein, 1.30 per cent.; sugar, 6; 27 ounces daily; calories, 65 per pound; gas, pain; loose green, yellow stools; given dextrinized wheat gruel, skimmed milk and maltose (dextri-maltose).

DATE:	Feb.27	Mar.5	Mar.10	Mar.17	Mar.30
Percentages: F. ..	.60	.60	.60	.75	1.00
P. ..	1.30	1.50	1.75	2.00	2.00
C. ..	6.	4.	5.	5.	6.
		WEIGHT			
Pounds	8	$8\frac{1}{4}$	$8\frac{2}{3}$	9	$9\frac{1}{2}$
		QUANTITY			
Ounces	32	32	36	40	40

NOTE: Sugar kept low because of sugar intolerance.

Age at termination of treatment, 1 1/7 month; duration of treatment, $4\frac{1}{2}$ weeks; gain, $1\frac{1}{2}$ pounds.

CASE 4. *History.*—L. H.; 5 months; birth weight, $7\frac{1}{2}$ pounds; present weight, 8 pounds; average for age 15 pounds; nursed two months; fed on varying proportions of milk, water and cream; now gets 2.65 per cent. fat; 1 per cent. protein; 5 per cent. sugar; 46 ounces daily; calories, 71 per pound; hard, crumbly, light, clay-colored stools; cries continuously; given dextrinized wheat gruel, skimmed milk and a semi-solid malt extract, boiled for a moment.

DATE:	May 30	June 5	June 12	June 21	July 4	July 14	Aug. 12	Sept. 11
Percentages: F.	.50	.50	.60	.80	1.00	1.25	1.50	2.00
P.	1.25	1.50	1.75	2.00	2.25	2.50	2.75	3.00
C.	6.00	7.00	7.50	7.50	7.50	7.50	7.50	7.00
			WEIGHT					
Pounds	$8\frac{1}{3}$	$8\frac{3}{4}$	$9\frac{1}{3}$	10	$10\frac{1}{2}$	$11\frac{1}{4}$	$13\frac{1}{3}$	$15\frac{1}{4}$
			QUANTITY					
Ounces	48	48	48	50	56	56	56	56
Calories:								
Per pound.....	65			75		75		

Age at termination of treatment, 8 months; duration of treatment, $3\frac{1}{3}$ months; gain, 7 pounds.

CASE 5. *History.*—M. A.; 8 weeks old; birth weight, 5 pounds; present weight, 5¾ pounds; average for age, 11 pounds; never nursed; light yellow, dry, hard stools; regurgitation much (main cause of loss of weight); fed upon milk, cream and water mixture; now gets fat, 2.50 per cent.; protein, 1.40 per cent.; sugar, 6 per cent.; 24 ounces daily.

DATE:	Mar. 12,	18,	25.	Apr. 1,	7,	14,	23,	30.	May 7,	14.
Percentages: F.	.50	.50	.50	.60	.70	1.	1.10	1.25	1.50	1.75
P.	1.40	1.50	1.50	1.50	1.65	1.75	1.75	1.75	1.75	1.75
C.	6.	6.50	7.	7.50	7.50	8.	8.	8.25	8.25	8.25
				WEIGHT						
Pounds	5¾	6	6 3/16	6 9/16	6 15/16	7¼	7¾	8¼	8½	9
				QUANTITY						
Ounces	24	24	24	28	30	30	32	36	36	36
Calories:										
Per pound.....	48					65		70		

Age at termination of treatment, 4 months; duration of treatment, 2 months; gain, 3¼ pounds.

CASE 6. *History.*—B. W.; 4 months; birth weight, 7 pounds; present weight, 8 pounds; average for age, 14 pounds; nursed two weeks, then weaned because of green stools; has had proprietary foods and milk mixtures; now taking: fat, 3.75 per cent.; protein, 2 per cent.; sugar, 4 per cent.; constipated; light colored stools, alternating with loose, curdy ones; given skimmed milk, dextrimaltose and water.

DATE:	May 3	May 5	May 9	May 25	June 26	June 29
Percentages: F.....	1.	1.	1.	1.25	1.35	1.50
P.....	1.30	1.50	1.60	1.75	1.75	1.75
C.....	6.	6.	7.	8.	8.	8.25
			WEIGHT			
Pounds.....	8	8⅓	8¾	9	9½	9¾
			QUANTITY			
Ounces.....	35	36	36	42	42	42

DATE:	July 1	July 15	July 21	Aug. 6	Sept. 16	
Percentages: F.	1.75	.50	1.	2.	2.	
P.	2.	1.	1.50	2.	3.50	
C.	8.25	3.	5.	7.	7,	
	WEIGHT					
Pounds	10¼	9½	9	10	13	NOTE: Reduced weights 7-1 to 7-21 due to acute diarrhea.
	QUANTITY					
Ounces	42	28	32	42	48	

Age at termination of treatment, 8 months; duration of treatment, 4 months; gain, 5 pounds.

CASE 7. *History.*—M. H.; 3 months; birth weight, 5 pounds; present weight, 7 pounds; average for age, 12½ pounds; condition good; partly nursed and also a mixture representing fat, 3.00 per cent.; protein, 1.33 per cent.; sugar, 6 per cent.; 4 ounces after fifteen minutes of breast; very hard, dry movements, of good color; no gain for several weeks.

DATE:	Apr. 4	Apr. 8	Apr. 12	Apr. 22	Apr. 30	May 12
Percentages: F.....	1.75	1.75	1.75	1.75	1.85	2.
P.....	1.50	1.50	1.60	1.75	1.75	1.75
C.....	7.	7.	7.	7.	7.	7.
			WEIGHT			
Pounds.....	7	7½	8⅝	9⅜	9 13/16	10⅜

			QUANTITY			
* *Breast*—						
Ounces.....	3	3-3½	3-3½	3-3½	3-3½	3-3½
Bottle—						
Ounces.....	21	21	21	24	28	28

NOTE: This case does not properly belong to cases treated in this paper; is included to illustrate gain under low fat after high fats had failed.

* Amount taken approximately at each nursing verified by weighing.

Age at termination of treatment, 4⅓ months; duration of treatment, 5½ weeks; gain, 3 pounds, 6 ounces.

CASE 8. *History.*—D. L.; 7 months; birth weight, 6 pounds; present weight, 10 pounds; average for age, 17½ pounds; nursed six weeks, then put on proprietary foods and milk mixtures; movements green, with large fat curds; emaciated, constantly lamenting and sleepless; given skimmed milk, flour and semi-solid malt extract boiled together.

DATE:	Nov. 8	Nov. 11	Nov. 15	Nov. 20	Nov. 25
Percentages: F.....	.50	.50	.60	.80	.90
P.....	1.25	1.35	1.75	1.85	2.
C.....	5.	6.	7.50	8.	8.
			WEIGHT		
Pounds.....	10	10	10¼	10¾	11¼
			QUANTITY		
Ounces.....	33¼	33¼	40	40	40
Calories:					
Per pound.....	36		51		

DATE:	Dec. 2	Dec. 8	Dec. 14	Dec. 27	Jan. 3	Jan. 17
Percentages: F.....	1.	1.18	1.28	1.40	1.75	2.
P.....	2.30	2.50	2.90	3.	3.25	3.25
C.....	8.25	8.25	8.25	8.	8.	8.
			WEIGHT			
Pounds.....	12	12¾	12½	13½	14	15
			QUANTITY			
Ounces.....	40	40	42	42	42	48
Calories:						
Per pound.....		64				

Age at termination of treatment, 9⅓ months; duration of treatment, 2⅓ months; gain, 5 pounds.

CASE 9. *History.*—R. G.; six months; present weight, 10 pounds; average for age, 16¼ pounds; never nursed; top milk mixtures; always regurgitated freely; stools good; now taking: fat, 2.25 per cent.; protein, 1.40 per cent.; sugar, 7 per cent.; given skimmed milk, dextri-maltose and dextrinized flour gruel.

DATE:	Aug. 12	Aug. 16	Aug. 20	Aug. 26	Sept. 1	Sept. 8	Sept. 15	Sept. 26
Percentages: F. ..	1.	1.	1.	1.10	1.25	1.50	1.75	1.75
P. ..	1.50	1.75	2.	2.25	2.50	2.75	3.	3.
C. ..	7.	7.	7.30	7.30	7.30	7.30	7.30	7.30
				WEIGHT				
Pounds	10	10¼	10½	10⅞	11½	12	12½	13
				QUANTITY				
Ounces	42	42	42	48	48	50	50	50

Age at completion of treatment, 7¾ months; duration of treatment, 6 weeks; gain, 3 pounds.

CASE 10. *History.*—Baby S.; never nursed; weight, 11 pounds; fed upon various top-milk mixtures; stools usually hard, dry and light, now acid and fermented; no regurgitation; is taking: fat, 4.85 per cent.; protein, 1.77 per cent.; sugar, 6 per cent.

DATE:	July 2	July 10	July 17	July 24	July 31	Aug. 14	Aug. 28
Percentages: F.....	1.	1.	1.	1.25	1.50	1.75	2.
P.....	1.75	2.	2.	2.50	2.75	3.	3.25
C.....	5.	6.	7.50	7.50	7.50	7.50	7.50
			WEIGHT				
Pounds.....	11	11¼	11½	12	12½	13	13½
			QUANTITY				
Ounces.....	42	42	48	48	48	48	48
Calories:							
Per pound.....							66

Age at termination of treatment, 8 months; duration of treatment, 2 months; gain, 2½ pounds.

CASE 11. *History.*—Baby T.; 6 months; birth weight, 6¾ pounds; present weight, 11 pounds; average for age, 16¼ pounds; never nursed; fed upon various proprietary foods; now taking condensed milk 1-8; very constipated; hard, brown movements; restless and sleepless; given skimmed milk, dextrimaltose and flour gruel, dextrinized.

DATE:	July 14	July 21	July 28	Aug. 10	Aug. 30	Sept. 20	Oct. 10
Percentages: F....	.50	.50	.60	1.	1.50	1.75	2.00
P....	1.25	1.45	2.00	2.50	3.00	3.25	3.25
C....	8.	8.	8.	8.	7.	8.	8.
Calories:							
Per pound....							64
			WEIGHT				
Pounds.....	11	11½	12	12⅝	13⅝	14¾	16

Age at termination of treatment, 9 months; duration of treatment, 3 months; gain, 5 pounds.

It is not claimed that high protein or low fat values succeed with all babies of this class. Although most of the cases here reported had a successful issue, we have had many failures; but infants of the malnourished type seem, in most cases, to thrive better if given low amounts of fat and comparatively high protein. An attempt should always be made to fit the food to the baby; to educate it to tolerate a rational dietary.

127 South Illinois Avenue.

LOW FATS AND HIGH PROTEINS IN INFANT FEEDING

PERCIVAL J. EATON, A.M., M.D., AND EDWARD B. WOODS, M.D.
PITTSBURGH

At the 1912 meeting I presented a short paper entitled "Problems of Infant Feeding, Illustrated by Cases and Charts," bringing to your attention the seemingly good results of low fats and high proteins in cases of artificial feeding in infants. Dr. Woods and I have continued this general plan of feeding in all suitable cases, and to-day bring you a series of cases and charts from one of our hospital services, showing what can be done with infants whose heredity is not of the best, and whose home environment has been bad. These infants were uniformly in very poor condition, and are typical of the feeding cases admitted to the Children's Hospital in Pittsburgh. Just as much care was taken to specify hours for feeding and the necessity of living up to all written directions as in private practice, and a printed form was filled out for each case, a new one being supplied with each change of formula. Certified milk and gravity cream from certified milk were alone used. No formulas were pasteurized or sterilized. Mead's dextri-maltose was used in each case and a proper quantity of citrate of soda (C. P.) (about 1 grain to each ounce of milk and cream in the mixture).

CASE 1.—H. R. (Hospital No. 4849), 6 months of age, was admitted because of a cough, diarrhea, and loss of weight. Family history negative, further than that the family were low-class Jewish people. On admission the child was found to have a suspicious ear, and the drum was punctured by a visiting aurist, and discharged pus for some days. The child was put on barley water for a few days and its gastro-intestinal tract was well cleared out. Then defatted milk was added in increasing proportions, and finally it was put on modified milk, which from time to time was increased in amount and strength, as the conditions of the child demanded. Because of the apparent good it had done, the barley was continued in the series of formulas, which were as follows:

Formulas	1	2	3	4	5
Fat	1.70	2.5	2.3	2.3	2.3
Protein	1.50	1.66	1.66	1.71	1.85
Amount in ounces...	36	38	42	49	49

His weight-sheet shows such a marked drop, and such a sharp rise during the second week, that we question whether the weight was correctly recorded.

CASE 2.—D. W., Hospital No. 5009. Six weeks old. No history. Pale, emaciated. Was kept in the hospital one month and discharged in good condition, with a gain of 3 pounds.

Formulas	1	2	3
Fat	2.2	2.3	2.3
Protein	1.6	1.7	1.75
Amount in ounces	28	35	42

CASE 3.—M. A., Hospital No. 4757. No history. Came in under my predecessor, and had been given whole milk mixtures until I came on duty the last

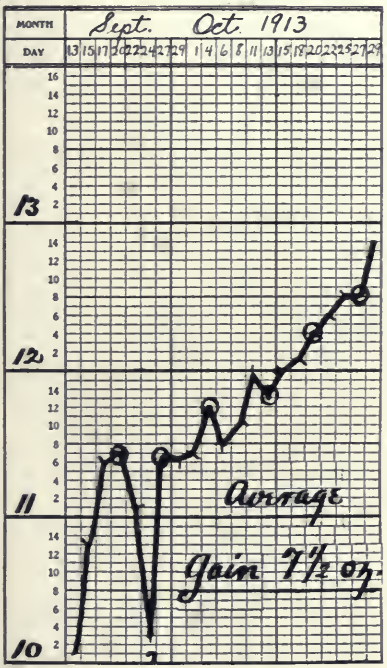

Fig. 1.

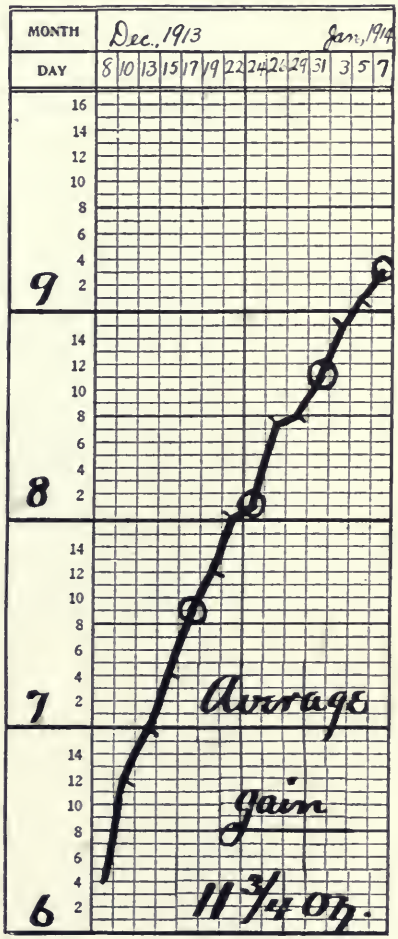

Fig. 2.

Fig. 1.—Weight record in Case 1, H. R., aged 6 weeks, Hospital No. 4849.
Fig. 2.—Weight record in Case 2, D. W., aged 6 weeks, Hospital No. 5009.

week in September. The contrast between the weight results of the two methods of feeding is striking. (Whole milk mixtures and percentage modification.)

Formulas	1	2	3
Fat	2.4	2.3	2.3
Protein	1.75	1.78	1.8 plus
Amount in ounces	40	49	56

CASE 4.—W. M., Hospital No. 5026. Three months old. Mother died seven days after child's birth of peritonitis. Was fed on condensed milk for two months, then Horlick's and, not liking that, on barley broth, which it did not

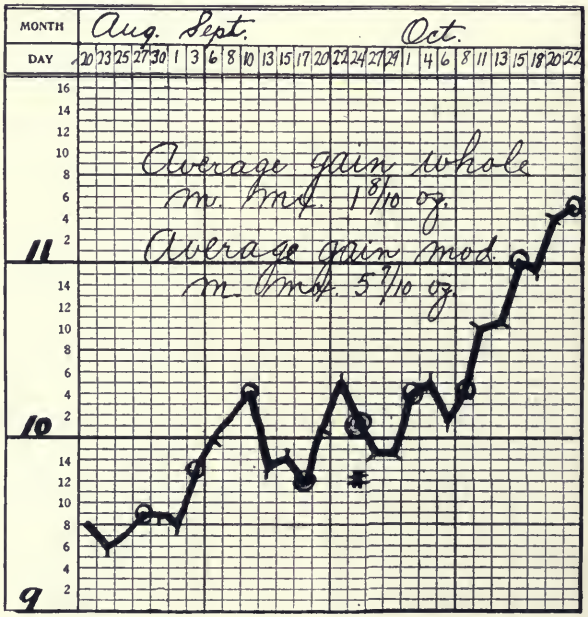

Fig. 3.—Weight record in Case 3, Mary A., Hospital No. 4757.

take well. For two weeks previous to admission to hospital was given certified milk and water, equal parts. This was vomited. General condition on admission was poor. Fontanelles widely open. Was discharged in good condition.

Formulas	1	2	3
Fat	2.	2.2	2.
Protein	1.4	1.5	1.62
Amount in ounces	35	49	54

The latter formula was given December 29 and was continued by my successor, Dr. Edwards.

CASE 5.—H. S., Hospital No. 4863. Ten months old. Has never been well. Various foods. Condensed milk for the last five weeks. Diarrhea for a week. Vomits some. For ten days after admission the child was given barley

water, and barley water with an equal volume of skim milk, and toward the end of this period whole milk and barley water about equal parts. September 29 the child was put on a formula.

Formulas	1	2	3	4
Fat	2.	...	....	2.3
Protein	1.7	1.7	1.86	2. plus
Amount in ounces.....	49	47	47	47

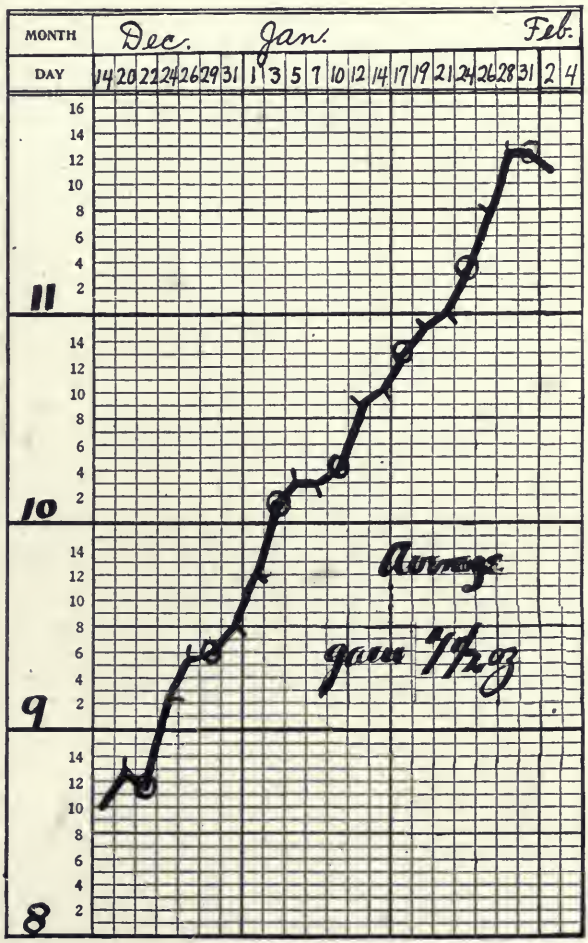

Fig. 4.—Weight record in Case 4, W. M., aged 3 months, Hospital No. 5026.

Barley jelly was added to each feeding in increasing amounts, and a short time before the child was discharged a raw egg was given, a half in each of two feedings. From October 15 for a couple of weeks the milk and cream were

boiled three minutes. Bulgara tablets were also given rather freely, as well as some iron. This case shows very clearly the difficulties which often, in hospital practice, seem insuperable. It further shows what persistence in rightly directed efforts may accomplish. The child left the hospital rosy and well.

CASE 6.—F. H. Jr., private patient. This case has been selected both for some of the lessons which a study of the feeding methods give, and also because the case is typical of what may be accomplished when the patient receives every care from the mother and nurse, and when one is sure that orders are carried out with exactitude.

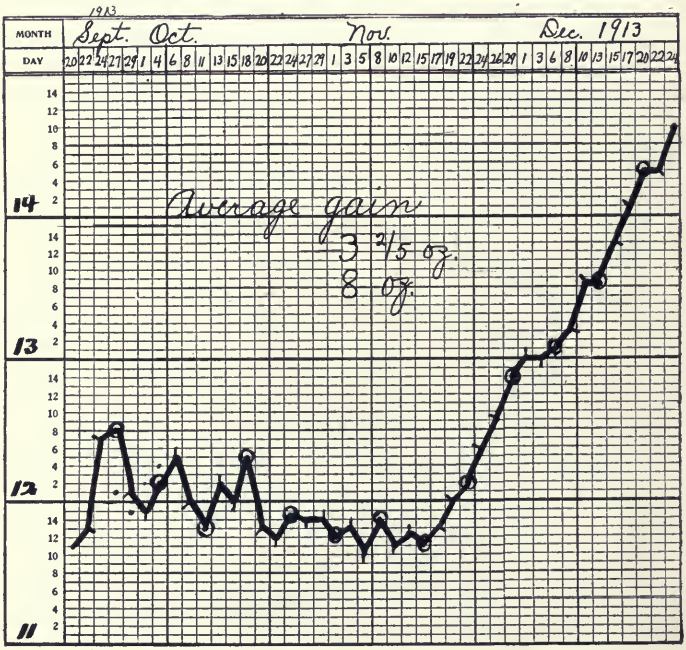

Fig. 5.—Weight record in Case 5, H.S., aged 10 months, Hospital No. 4863.

The child was first seen when 10 weeks old. It had gained very little since birth, and its weight had been variable, but no record had been kept. It was fussy and troublesome; had been on some high fat formulas, and had promptly had an attack of fat poisoning. Quite a number of other foods had been tried. It was put on a raw milk formula of properly proportioned fat and protein with citrate of soda and dextri-maltose, and immediately began to gain. It had an uninterrupted season of growth and contentment up to the eruption of its first teeth, when it was somewhat upset.

Formulas	1	2	3	4	5	6	7
Fat	2.	2.2	2.3	2.3	2.	2.3	2.3
Protein	1.47	1.57	1.66	1.8	1.87	2.	2.3
Amount in ounces...	32	36	42	49	56	56	56

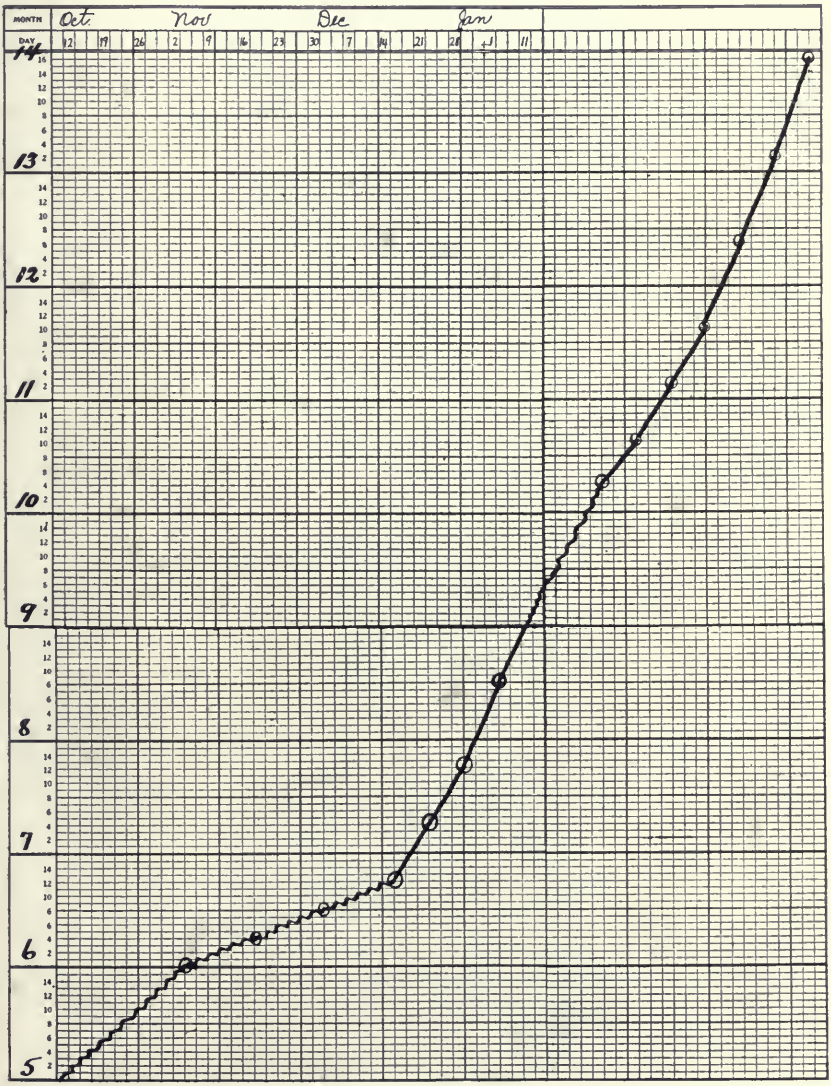

Fig. 6.—Weight record in Case 6, F. H., Jr.

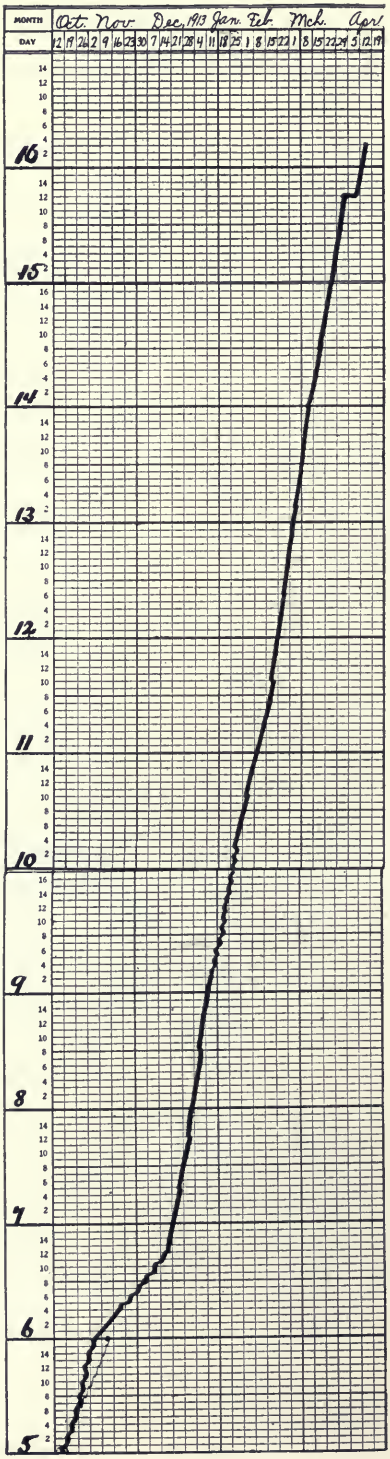

Fig. 7.—Record of weekly gain in weight of infant, F. H., Jr.

Cereal jelly was added to each feeding in Formulas 5, 6 and 7. Its first weight-chart is made up from three weighings per week, and its second is a record of its weekly gain.

We wish to emphasize some of the points made in our paper of two years ago:

The first, and perhaps the most important, is that even in hospital work, babies fed after this general plan are less inclined to be ill, and have a more stable life, as well as a very consistent growth.

Second. The formulas should be adapted in quantity and quality as closely as possible to the individual infant.

Third. These formulas, and all that we use now-a-days, are practically home-modified mixtures, which Bowditch and Bosworth have shown are more nearly correct as to percentages than are laboratory mixtures, at least as a rule.

Finally we desire to call attention to the printed form which has been referred to earlier in the paper, as having been filled out each time a new formula was given, whether in hospital or private practice. It is so simple and so complete that we rarely find nurse or mother so stupid as to be unable to carry out its directions.

Feed at Date,

Remove all the cream from ... qt. "Certified" Milk, or from FRESH, RAW MILK which has stood in bottle, on ice, for 10 hours. Stir this cream.

Cream,	ounces.	Divide among ..... feedings
Midmilk	ounces.	
Water,	ounces.	**Note:**
Limewater,	ounces.	
"Solution,"	teasp.	
Sugar of Milk,	tablesp.	
	teasp.	
Dextri-Maltose	tablesp.	
	teasp.	

(F....P....) Always Bring This Card.

DISCUSSION ON PAPERS OF DR. MILLER, AND DRS. EATON AND WOODS

DR. TALBOT: I would like to ask Dr. Miller how he explains why some babies taking raw milk have casein curds in their stools, have symptoms of indigestion, and do not do well; and, taking the same formulas, but boiling the milk sufficiently, the babies do well and have no curds in their stools. If I understood his paper correctly, he said that protein could not do any harm.

DR. MORSE: It seems to me that these two last papers and the way they have been presented as methods of infant feeding are distinct steps backward. It seems to me that in this country we have got by the time of talking about a method of infant feeding with low fat and high protein, or with high fat and low protein, etc. Anybody that has had any large experience in feeding can bring out a series of cases that have done well on low fats and high proteins; anybody can bring out, also, a series of cases that have done badly on the same.

The same way with the sugars. It seems to me this is a step backward, that we ought to feed our babies individually according to indications in each case, as shown by the history, by the symptoms, by the examination of the stools, and what they do on the food that they are given as an experiment. The food must be fitted to the individual baby. There can be no method of feeding that is generally applicable.

DR. JACOBI: I wish simply to say that I totally disagree with the remarks of the last speaker.

DR. MORSE: That has always been so, Doctor.

DR. JACOBI: And if the Doctor will not improve, it certainly will always be so. General rules are not made for individuals. Rules are made for our general population or adults or children. There is an average of healthy babies, healthy children, healthy adults; and the general rules that should be followed in the feeding and in the law-giving generally must be an average. So it has been. I know that Dr. Morse speaks from experience. So do I; my experience in the last fifty or sixty years has shown me that the babies and the patients generally are better off when guided by a general rule followed by a nurse or doctor with little knowledge and much common sense.

I am not of the opinion that every case must necessarily be studied individually from the very beginning. A case should be studied individually only when that baby or adult does not thrive when treated according to a rational general rule. Then the shrewdness, the knowledge, the brightness of the doctor are required; but as a rule, as all of us are guided in our principles and politics by an average rule, and do well with it, so average rules do well in the feeding of the infant and of the child.

Now I am not of the opinion that we must not or can not feed a baby before that baby has been studied from top to toe and from behind forward; that is impossible, and it is not necessary; and those remarks that have been made in the last paper covering that truism are correct. The question is whether that is still necessary to repeat. It appears to me that a great many of these papers need not be written; and if old papers were minded, for instance my own, (laughter and applause), I am still of the opinion that in connection with what you have heard in the last paper and in connection with what you were taught forty and fifty years ago which has gone over the country, and been appreciated by a great many practitioners, they are still correct. If a case turns up that can not be guided by old, simple rules, then it is time to study that one case very intimately. That baby is either imperfect, or it is sick. Sometimes even send it to the laboratories, if you can not determine formulas yourself. So that is my opinion as to the remarks of the last speaker, whom I have had a great many opportunities to admire, and a great many opportunities to find fault with.

Now, so far as the remark is concerned that raw milk and boiled milk do not behave in the same way in connection with the appearance of the feces, I think that is an old story, too. Those of you who preferably read German books and French journals in place of American books—there are so many among you who have that failing—will remember to have seen an article lately, only six or eight weeks ago, in the *Journal für Kinderkrankheiten,* in which a German makes the discovery that raw milk will always result in curdy stools, and cooked milk doesn't. That is not a new discovery except for the author. That discovery has been made a hundred thousand times by mothers, and now and then, even by doctors. I was never charged with being funny or witty—it is not in my line; but I want to refer you, as one of the predecessors of that German who has made that discovery, to a colleague in Chicago; I don't remember just now whether it was Helmholtz or Brennemann, or someone else. You

may have seen his article in which he makes just that point, that the stools will curdle when the milk is raw; that they will not do so when the milk is cooked. Now that will stand. It has been standing a long, long time. It does no harm, evidently, even in this learned circle, to repeat these things. It is good that such questions come up again; for babies will turn up again, all the time, and audiences change, and the babies suffer from our ignorance and our forgetfulness. What the man in Germany and the man in Chicago and men before him said I repeat now. I think we should study our own literature a little; and if your future articles are not long and fatiguing, perhaps a short article will do just as well or better.

DR. CHAPIN: The pendulum has swung far in our infant feeding. A few years ago it was all high fats. It has seemed to me it has swung a little too far in the other direction; that is, toward low fats. There is no doubt that in institutional life children do not digest fats very well and the formulas that have been given are, perhaps, on the whole better; but the way to get over that is not to keep the children in the institutions. They need fresh air to oxidize their fats; and I feel, when we go all over the country and see institutions being put up for infants, we can be pretty sure that they are going to be low-fat infants because they can not digest the high fats. They need individual care and fresh air to oxidize their fats; and I think that from the teaching of this exceedingly low-fat percentage we are going to see a crop of rickets in the future. We have woman's milk to imitate; and we know that the average milk given by the woman to her baby is not so very low in fat; food principles are not interchangeable. That is one of the disadvantages of the overemphasis on the caloric system. The baby must have individual care and plenty of fresh air and mothering to digest fats. So far as institutions are concerned, I believe these papers are true.

DR. JACOBI: If I may add one more word I will repeat here what I have said a hundred times. "Burning" stools are the result of raw milk, and of sugar. Many of you will insist on using large quantities of milk-sugar. If you do that, you will have the burning stools. Not in every baby; indeed, it is impossible to kill every baby. If you will insist on using milk-sugar exclusively, you will not infrequently have diarrhea and burning stools; and I preach again —and I have done it, and I have not been obeyed. I shall not be minded any more to-day than I have been minded these fifty years—use cane-sugar and your babies will be better off. There is sufficient milk-sugar for digestive purposes in all of the milk combinations that you will use, and all the rest is injurious. The burning stools are the result of the large amount of milk-sugar and the use of raw milk.

DR. HOLT: Dr. Eaton has given us some interesting weight-charts from his hospital experience. I would like to ask if they represent his average results or if they are selected cases?

DR. KNOX: Dr. Miller's paper corroborates my own experience with this group of cases, the convalescent cases in the summer after intestinal disease. I have found the form of milk which we could give most safely at this time was skim-milk, and that, added to the cereals in small quantities at first, increased to larger proportions, up to half and half. This brought the child forward in convalescence, it seemed to me, with fewer setbacks than any mixture of *whole* milk. I have rarely seen in any of these cases the development of intoxication which I could attribute to sugar; exceptionally has sugar been found in the urine, nor have I seen starch with the iodin test in the feces, even after a strong cereal mixture; so that the old method which has come back to

us from experience of the past still seems valuable in the treating of these convalescent cases, namely, water, then weak cereal, and to this the addition of small and gradually increasing quantities of scalded skimmed milk or, more lately, protein milk.

DR. DUNN: I think that the whole history of infant feeding must bear out Dr. Morse's ideas that it is the factor of individual variation which has to be the main thing in infant feeding.

In the very beginning, the difficulties of artificial feeding were attributed to the relatively high percentage of casein in cows' milk as compared with human milk; and all our earlier methods of modifying milk were directed at the curd. Such methods as the addition of alkalies, sodium citrate, and other substances; as the addition of cereal diluents to break up the curd; as the use of whey, to try to reduce the proportion of casein to soluble nitrogenous content; as the use of peptonization, these were each advocated at some time as the one method of feeding infants which would transcend all others; and always at the times when those measures were advocated, a series of cases were reported that apparently gave just as good results as those now reported with low fats and high proteins. Then came a period in which other elements of the milk were regarded as the principal deleterious factors, when the lactose was regarded as the harmful element of cows' milk and when it was advocated that we should always change milk-sugar to maltose or some other sugar. Then came a period in which just the reverse of our older ideas came up; the casein was considered perfectly harmless and the trouble in infant feeding was attributed to the fats, or the sugars, or the salts.

Every one of these so-called feeding methods *worked*—every one of these ideas in infant feeding has had a certain amount of experimental work or careful observation to back it up; and every one has apparently shown good results in certain series of cases. I think that we can not do otherwise than to accept these various views as being partial glimpses of the truth. It seems to me that it is quite true that a large number of babies do show the individual peculiarities of not being able to tolerate a high percentage of fats, and being able to tolerate high percentages of proteins. So much is that true, that in cases of a certain type at the Infants' Hospital, we are accustomed, where we can not draw satisfactory conclusions from the history and have no examination of the stools or other evidence to go on, to begin our treatment by giving them low fats and high proteins, very much after the method described in the two papers that have been read. We do it because, as Dr. Jacobi says, until we have evidence to go on, we have got to feed our baby on something, and we have to select that method of feeding which we think will be most applicable to that case.

I don't think, however, that we should entirely lose sight of past conclusions. It seems to me that all of the various measures of modifying milk have been wrongly hailed as *methods* of feeding babies, and people talk about the method of feeding babies with low fats and high proteins, or some other method. I think speaking of the various ways of modifying milk as feeding methods is, just as Dr. Morse says, a distinct step backwards in infant feeding, and that we should recognize that each of the known facts about infant feeding is simply a measure or resource to be used in certain cases. I believe, further, that we should hail each new scientific addition to our knowledge of digestion and nutrition, with its practical application, not as a new feeding method to supersede others, but as an additional resource or weapon in a problem whose difficulty is such that we need every available weapon and even more. Thus we have a great variety of different measures or weapons to use, each applicable to the individual case, and what we need and are working for now is finer

methods of diagnosis, so that we can meet individual variation correctly a little earlier.

DR. ABT: I quite agree that there is no method of infant feeding. We should not designate methods of feeding; each baby must be considered. It seems to me, however, especially those who are teachers, that we must formulate knowledge for ourselves, at least, and for our students, in such a way as to make our plan of teaching perfectly definite, and consider the feeding and the baby in all possible relations.

First of all, we must consider the baby and his tolerance for food; that is the individual problem. Then we must consider the food in its relations; how much it can do towards upbuilding the processes of the body and how much injury it may do. We ought to have some definite knowledge as to the injuries that could be produced by a certain kind of food, based on a knowledge of the physiology of the food, of the nutritional value of the food, and of its reaction in certain individuals. I think we have that knowledge up to a certain point. I think we know that up to a certain degree fats will be taken care of; that beyond this point, in some babies, fats will do harm. I think we know the same about sugars. So that it seems to me that we must study the food in its relation to digestion and metabolism and appropriation, and the damage or injury that may be done by the foods; and then we must study the baby in its broader relations. For example, some babies have constitutional aversions to food; or, some babies will under no circumstances digest normal or ordinary amounts of fat. Some babies will under no circumstances digest ordinary or normal amounts of sugar. Some babies are constitutionally, as I emphasized yesterday, and I think it will be noted in a paper to-day, have certain idiosyncrasies against fats, sugars and proteins, and so on; and it seems to me the babies must be studied in those relations. I believe that all our progress along feeding lines must be made in this direction; first of all studying the individual nutrition of the babies, the nutritional points in the physiology plus food, and studying the babies so far as abnormalities or anomalies in constitution are concerned.

DR. MILLER: Dr. Talbot's question as to curds—I don't know why—I don't think that it has been very satisfactorily explained why—boiling the milk causes disappearance of curds. I am sorry that I was unable to present the charts, because they would have given the substance of this paper much more graphically, and because it seems to have been misunderstood in many of its essential features.

As to sugar, I said that I had never seen any injury from sugars in infant feeding, except temporary ones that could be readily rectified. The child with too much sugar has numerous acid and burning stools, a condition, it seems to me, that is easily rectified by cutting the sugar out or down at once; and this may occur with any sugar. It certainly occurs very often on malt sugar; it occurs frequently with children taking Mellin's food, which is rich in maltose. As to the question of method, this was not presented as any method in infant feeding. I don't believe that there is any method of infant feeding, myself, but I don't know any other name by which to call it, in reading the paper, without speaking of the method. Dr. Morse, himself, must have a method of feeding; he tries to adapt the food to his babies' digestive peculiarities, and he probably begins with some with 1 or 2 per cent. fat, some with 1, and some with a lower or higher per cent. of proteins or sugar. If I had read all the reports of cases, some of the children would have been found to have begun with 1 per cent., some with ½, and some with only ¼ of 1 per cent. of fat. It would have been

observed, also, that the fat was always rapidly increased, after the manner of immunizing animals. My rule was, every sixth day to make an increase of 1/10 of 1 per cent. of fat or more, according to their amount of tolerance. Usually, in the course of five or six weeks, or longer, they would be taking from 2 to 3 per cent. of fat. If any child is kept indefinitely without fat, of course, it will probably develop rickets or other nutritional disorder, but these children never had the fat below 2 per cent. for more than six or eight weeks. This was the point of my paper: to feed low fats and high proteins at first; and then, as quickly as possible, to increase the fats, and get the children on a rational dietary. These marasmic or undernourished babies do better under this plan than they do on the old one of giving them moderately low fat and protein. This has been my experience.

Another point: every one of these children (there are eleven children treated of in this paper) were in the better walks of life. They were more or less in the open air; they had the advantages of the sea air constantly; most of them were born at the seashore; and, therefore, they are not to be compared to institution children, the condition being induced through faulty feeding, or some peculiarity in the child itself.

DR. EATON: In answer to Dr. Holt's question, I would say those were ordinary, average cases in the Children's Hospital. These were picked out in particular because they were among the most miserable of the children we had on our service.

Dr. Morse's criticism is a particularly good one. I didn't realize that I had used the word "method." I didn't in the first paper. I said "this general plan"; and I didn't use the word "method" in the summing up. If Dr. Morse will remember, in the paper which I presented two years ago, I reported, I think, six or eight cases in private practice fed on this general plan, and in my conclusion I made a most insistent plea for recognition and study of the needs of the individual infant and the adaptation of the formula to each child; and I referred again, in this paper, to this conclusion. The formula should *always* be adapted, in quantity and quality, as closely as possible, to the needs of the individual infant. I believe that one's environment has a good deal to do with the trend of one's thoughts and one's practice. In my own part of the country—in my city particularly—I have to combat the treatment of children by a physician from a distance who treats children there, and quite a large number of them—by telephone, and who prescribes very high fat formulas. I don't think that needs any explanation, but I do have more of that to combat than probably any of you do; and that is the reason why I insist on the individual adaptation of the formulas. I do again wish to call particular attention to the fact that children so fed do well, and particularly well in the matter of not having what the laity call bilious attacks, which, in my experience, are very largely attacks—in Pittsburgh, at least—of fat poisoning. I also wish to insist that in hospital work, as well as in private practice, these infants, which are as a rule, all miserable cases, do better on the whole on mixtures of modified milk containing, comparatively speaking, a high protein content and a low fat content.

I most thoroughly agree with what has been said by Drs. Morse and Jacobi, that the things of life that fall into great classes, great groups, should not be treated by any one "system" or "method," to use those two words. I dislike those words as much as Dr. Morse does. I believe in giving each individual child that which seems best adapted to its needs, and that is as far as I can go in any discussion of infant feeding.

THE CHEMICAL COMPOSITION OF DIARRHEAL AS COMPARED WITH NORMAL STOOLS IN INFANTS*

L. EMMETT HOLT, M.D.
ANGELIA M. COURTNEY AND HELEN L. FALES
NEW YORK

It has been a matter of considerable surprise on going through medical literature to see how little has been written regarding the chemical composition of diarrheal as compared with normal stools. Stools of all varieties have been studied in many ways in great detail since the work of Uffelmann and Wegscheider about forty-five years ago. Numerous reports have been published of metabolism work which contain stool analyses made with reference to the different constituents, fat, nitrogen, ash, etc.; and scattered through these are found reports of isolated instances in which very loose stools have been studied, but, so far as we are aware, no attempt has been made to correlate the observations on diarrheal stools and compare them with those which were normal. The work done on diarrheal stools as such has been chiefly a study of the flora.

It was with the belief that some new light might possibly be thrown on the treatment of such conditions, dietetic and otherwise, that we have made from another point of view a restudy of our metabolism observations, namely, to determine the actual chemical composition of diarrheal stools and compare them with the normal.

Those whose observation on infants' stools is limited to an inspection of napkins can form but an imperfect idea of even the gross condition of the infants' excretions, especially as to the amount of water which they contain. When stools are received in a metabolism basin a very different impression is made.

With the two large factors in the production of loose stools, that is, increased secretion and increased peristalsis, every one is familiar. But many questions arise relating to diarrheal stools. Do very loose stools differ from normal ones only in that they contain more water, or do they actually represent a much greater loss of solids? Are all

*From the Laboratories of the Babies' Hospital and the Rockefeller Institute.

the solids in the stool affected to the same degree, or are some drained away much more than others? Does the actual loss, or the percentage of intake lost, vary only with the food, or are there other important factors?

These were some of the questions regarding which it was thought information might be gained by such a study as that undertaken. The data have been derived by going through the records of our metabolism studies for the past four years and selecting from them the cases illustrating the different types of stools. Such records were especially valuable for our purpose, for, although the children studied differed considerably with respect to their condition and the food given, the exact intake of the different food elements had been determined, as well as the composition of the excretion. In a few of the early cases the stool analysis did not go further in the ash estimation than to determine the total ash; but in all the recent ones, including the greater number studied, the composition of the ash had been fully worked out.

While the chemical composition of the stools was much influenced by the food, it did not seem to make much difference what the exciting cause of the diarrhea happened to be, as its effect in draining away fluids and solids from the body was much the same in all cases studied.

For the purpose of this study the cases were divided into (A) formed, firm or pasty stools; (B) loose stools and (C) very loose stools. The division into loose and very loose is a somewhat arbitrary one. We classed as "loose" those in which the total quantity of water lost varied between 100 and 200 c.c. daily; as "very loose" those in which the water exceeded 200 c.c. daily.

No account was taken of the number of evacuations during the day, first, because in metabolism observations this is somewhat difficult to determine, and secondly, for the reason that it is really of little importance. What is essential is the total daily excretion.

In the group A, of stools of normal consistency and appearance, there were observations made on 9 patients for 11 periods. In the group B, loose stools, there were observations made on 8 patients for 14 periods. In the group C, very loose stools, observations were made on 6 patients for 10 periods. In all, observations were made on 21 patients for 35 periods, for a total of 128 days.

A different period signifies an observation on the same child at another time or on another food. In all but two instances each period

was for a longer time than a single day; usually it was three or four days. This was found convenient and sometimes necessary, since in estimating some of the salts of the ash, of which the quantity was very small, the total secretion of two or three days was needed for a satisfactory quantitative determination. The amounts of the substances given in the table represents an average for the period. Two or three days were put in the same period only when the food was the same and the stools similar in character. In all cases daily averages of all the observations are given in the charts, and in several cases we have given the range. The extreme variations were considerable, but not greater than one would expect and not enough to affect the general significance of the findings.

The type of child studied is indicated in the clinical summary of cases appended (Table 1).

The average age of the twenty-one children was 9 months (the range being from 4 to 13 months) and the average weight 5,500 gm. (about 12½ pounds). In general, they would be classed as hospital nutrition cases. Those in Group A, however, were digesting well and gaining satisfactorily in weight; those in Groups B and C were suffering from acute, or more often subacute diarrhea, usually of a non-febrile character. There were no dysenteric cases and none of acute infection or intoxication of the intestines of a severe type.

It will be seen from the table that most of the infants were taking formulas derived from whole milk by various dilutions. This was true in twenty of the thirty-five periods studied. The exact percentages of the milk formulas are given in the table. The numerals indicate the fat, sugar and protein, given in order. Thus, in Case 1, Group A, the food was fat, 2.4 per cent.; sugar, 5 per cent.; protein, 1.8 per cent. The sugar added was lactose unless otherwise specified. In sixteen periods some preparation of maltose was given. Usually this was a fluid preparation, a mixture of maltose and dextrin.

The protein milk (*Eiweissmilch*), which was the food in five periods, was made from whole milk and had the following average composition: Of seven samples, fat 3.00; sugar 2.90; protein 4.00; ash 0.70. It was prepared in 10-quart quantities by precipitating 4 per cent. milk with rennet; the curd was carefully washed twice with water and then rubbed through a fine wire sieve with the addition of 10 pints of special buttermilk (fat-free lactic acid milk) and water added up to 10 quarts.

TABLE 1.—Clinical Summary of the Cases Studied

GROUP A (STOOLS OF NORMAL CONSISTENCY)

No.	Name	Age, Months	Weight, Gm.	Food	Period	Duration, Day	No. Stools a Day	Gross Appearance
1	P. L.	10	9,600	2.4; 5; 2.2. With barley flour	1	3	1-2	Brownish-yellow, largely formed, smooth, firm.
1	P. L.	10	9,600	Same as preceding.....	2	3	1-2	Like preceding.
2	D. W.	5	5,800	1.8; 5; 1.6	1	3	4	Dry, pasty, yellow-brown, not homogeneous.
2	D. W.	5	5,800	Same as preceding.....	2	3	2	Like preceding.
3	W. H.	12	7,000	2; 5; 1.8. With cereal gruel	1	3	3	Pasty, gray-green, mostly smooth, partly formed.
3	W. H.	12	7,100	Same as preceding.....	2	3	2	Mostly smooth, formed, gray, some softer, greenish.
4	F. H.	11	4,500	¾ protein milk; ¼ barley water	1	4	3-4	Mostly formed, soft, yellow-gray, smooth.
5	J. S.	7½	4,100	1.45; 4.5; 4. Made like protein milk	1	3	1-5	Mostly formed, yellow-gray, smooth.
6	A. R.	8	5,445	Protein milk with barley flour	1	3	3	Formed, dry, constipated.
6	A. R.	8½	5,695	2.4; 2.6; 2.2. With barley flour	2	3	1-2	Similar, but not so hard.
7	F. B.	4	5,745	Protein milk and rice flour	1	3	2-4	Pasty, smooth, largely formed, brownish yellow.
				GROUP B (LOOSE)				
1	D. W.	5	5,700	1.8; 5; 1.6. With malt.....	1	4	5	Watery, greenish, curds and sour.
2	J. D.	5	3,900	1.6; 2; 1.4. With cane sugar and barley flour	1	4	5	Partly thin, pasty; partly watery, granular, greenish.
3	V. C.	4	4,400	1.8; 2.2; 2. With barley flour	1	3	8	Watery, fine fat curds, sour, greenish-yellow color.
4	E. R.	10	7,500	1.8; 5; 2. With cereal.....	1	3	4-5	Watery, yellow, fat curds, mucus.
4	E. R.	10	7,400	Same as preceding.....	2	3	5	Partly watery, partly solid, green, sour, mucus.
4	E. R.	10½	7,300	1; 5; 2. With cereal gruel..	3	4	3-4	Watery, partly green, large curds and mucus, sour.

5	B. S.	12	8,200	2; 5; 1.8. With cereal gruel	1	4	3	Watery, gray-green, granular, foul.
6	W. H.	13½	6,700	2.6; 5; 2.4. With cereal gruel	1	3	4	Watery, yellow, fat curds and mucus.
7	F. H.	11	4,800	0; 4.5; 3.5. With cane-sugar	1	3	11 13	Orange-yellow, pasty, watery, and much mucus and hard curds.
7	F. H.	11	4,700	1.6; 4.7; 3. With cane-sugar	2	3	8-10	Watery and pasty, smooth, curds and mucus.
7	F. H.	12	5,200	2; 4.5; 2.6. With dextrimaltose and sodium phosphate	3	4	5-10	Watery, foamy, fat and hard curds and mucus.
8	J. G.	7½	4,400	2.4; 5; 2.2.....	1	4	4-5	Watery, fat and hard curds, large fragments.
8	J. G.	8	4,500	Same, plus 6 dr. maltose	2	3	4-6	Watery and soft, spongy, small curds.
8	J. G.	8	4,600	Same, plus 9 dr. maltose	3	3	3	Watery, brownish-yellow, hard curds, foul.

GROUP C (VERY LOOSE)

1	B. S.	12	7,700	2; 5; 1.8. With cereal.....	1	2	5-8	Very watery, green, with fat curds and mucus.
2	V. C.	4	4,700	1.8; 5; 2. With barley flour	1	4	6	Watery, green, with large fat curds; sour.
2	V. C.	4	4,500	1.8; 2.2; 2. With barley flour	2	5	7	Like preceding with mucus also.
3	C. B.	8	4,900	2.6; 5; 2.4. Olive oil 1½ dr.; changed to protein milk	1	5	5	At first pasty with fat curds, then green, very watery, with fat and hard curds and mucus.
4	F. H.	10	4,500	2.6; 5; 2.4	1	8	11	Watery, green, foamy, fat curds and mucus.
4	F. H.	10	4,600	2.6; 5; 2.4.....	2	9	9	Very watery, green, large fat curds. and mucus.
5	W. S.	5	4,600	1.6; 5; 1.4.....	1	2	6	Watery, greenish, little solid matter.
5	W. S.	5	4,600	Same as preceding, with 8 to 12 c.c. of malted milk	2	8	4-7	Watery, greenish, little solid matter.
5	W. S.	5	4,400	Fat-free milk and soy-bean gruel	3	1	7	Watery, greenish, little solid matter.
6	D. L.	9	5,200	1.8; 5; 2.1.....	1	1	10	Watery, green, with curds and mucus; sour.

The high ash content of protein milk shown by the analyses was to us a surprise, since the impression has prevailed that the salts have been much reduced by the removal of the whey and by the washing of the curd. These results, however, were based on seven complete examinations including thirteen samples, some of these being composite samples. Prepared in the manner described, the range of the salts in protein milk was from 0.54 to 0.74 per cent.

Table 2 gives the percentage of water and dried matter in stools of different types, showing both the averages and the range met with in the series.

TABLE 2.—PERCENTAGE COMPOSITION OF STOOLS STUDIED

	Stools of Normal Consistency		Loose		Very Loose	
	Seven Patients Eleven Periods		Eight Patients Fourteen Periods		Six Patients Ten Periods	
	Average %	Range %	Average %	Range %	Average %	Range %
Water	80.3	(73-85)	91	(88-92)	93	(90-96)
Dried matter..	19.7	(15-27)	9	(8-12)	7	(4-10)

METHODS EMPLOYED

Food and feces were each dried on a steam-bath to a constant weight in equilibrium with room temperature and humidity, and all the determinations were made on this dried material. Each of the urine values was obtained separately from liquid samples. The fat in both the food and the feces was determined by extracting with ether according to a modification of the Soxhlet method and weighing the extract.

Total nitrogen was determined in the dried material and in the urine by the Kjeldahl-Gunning method. We have in our tables regarded all the nitrogen as representing protein; with the knowledge, however, that there is here a small error, as other nitrogen compounds, not protein, are present both in the urine and the stools and possibly also in the food. The amount, however, is so small in the infant as to be negligible for our purposes.

Total ash in dried food and feces and in urine evaporated to dryness was determined in most cases by the Stolte method. According to this method a platinum dish containing the sample is set on pieces of broken pipe-clay inside a porcelain dish. Low heat from a large-sized Teklu burner is applied to the porcelain dish until the material is well charred, after which the heat is gradually increased to the greatest possible point. When most of the black has disappeared the platinum dish is covered by a piece of platinum foil and the heating continued until ashing is completed. No sodium or potassium chlorid is volatilized by this treatment. A few of the earlier samples here reported were ashed over a free flame with the addition of a few drops of acid.

TABLE 3.—Total Daily Excretion

	Stools of Normal Consistency			Loose			Very Loose		
	Seven Patients Eleven Periods			Eight Patients Fourteen Periods			Six Patients Ten Periods		
	Average		Range	Average		Range	Average		Range
Water (c.c.)	35.6	.....	(18. -50.)	133.	.....	(107. -195.)	293.	.....	(217. -555.)
Dried matter (gm.)	8.75	.....	(6. -13.1)	12.95	.....	(10.43 - 16.76)	21.16	.....	(12.6 - 37.35)
Fat (total)	3.12	.....	(0.91 - 7.07)	4.04	.....	(0.43[1] - 7.35[2])	7.97	.....	(1.39[3]- 12.95)
Neutral fat	.....	1.09	(0.38 - 2.08)	.....	2.83	(0.43[1] - 6.48[2])	.....	5.70	(1.37[3]- 8.24)
Free-fatty acids	.....	0.34	(0. - 2.04)	.....	0.72	(0.[4] - 1.44)	.....	2.44	(0. - 7.29)
Soap fats	.....	1.69	(0.46 - 5.40[5])	.....	0.50	(0.[6] - 1.91)	.....	0.74	(0.[7] - 2.17)
Protein	2.26	.....	(1.31 - 3.19)	3.56	.....	(2.42 - 5.76)	5.56	.....	(3.80 - 10.7)
Ash (total)	2.17	.....	(1.21 - 3.58)	2.72	.....	(1.47 - 5.93[8])	3.96	.....	(3.03 - 4.78)
CaO	.....	1.02	(0.46 - 1.70)	.....	1.08	(0.42 - 2.33)	.....	0.83	(0.70 - 0.96)
MgO	.....	0.096	(0.03 - 0.164)	.....	0.10	(0.025 - 0.15)	.....	0.12	(0.10 - 0.16)
P_2O_5	.....	0.63	(0.14 - 1.28)	.....	0.70	(0.12 - 1.53)	.....	0.59	(0.49 - 0.68)
Cl	.....	0.022	(0.008- 0.032)	.....	0.14	(0.05 - 0.37)	.....	0.28	(0.27 - 0.30)
K_2O	.....	0.180	(0.084- 0.300)	.....	0.36	(0.22 - 0.52)	.....	0.82	(0.68 - 0.97)
Na_2O	.....	0.041	(0. - 0.256)	.....	0.17	(0.11 - 0.31)	.....	0.36	(0.19 - 0.67)

1. Infant on fat-free milk.
2. Infant on milk containing 1-8 per cent. fat.
3. Infant on fat-free milk.
4. Two infants had none.
5. Very large stools, normal; infant gaining.
6. Six infants had none.
7. Two infants had none.
8. Fat-free milk; intake 9.86

Chlorin was determined in the urine by titration with silver nitrate, according to Volhard. In the dried material in most of the samples the extract, to which nitric acid and ferric alum had been added, was filtered after several hours' standing and titrated with silver nitrate. In the earlier cases the sample was charred, sometimes after being mixed with sodium carbonate, extracted with water, filtered and the filtrate was titrated with silver nitrate.

Phosphates in the urine were determined by titration with uranium acetate or nitrate, potassium ferrocyanid being used as indicator. In the dried material the Neumann method has been used in most cases, that is phosphoric acid precipitated in the ashed material as phosphomolybdate, dissolved by ammonia and reprecipitated by magnesia mixture as magnesium ammonium phosphate and weighed after ignition as pyrophosphate. In some of the earlier samples the Vozárik method was followed. According to this the material is well mixed with magnesium oxid and ashed, the residue dissolved in hydrochloric acid, sodium acetate and acetic acid added to the neutralized solution and phosphoric acid determined by titration with uranium nitrate. Calcium and magnesium, sodium and potassium were determined in dried material of food and feces and urine by the ordinary methods, calcium being precipitated as oxalate and weighed as oxid, magnesium weighed as pyrophosphate, sodium and potassium separated as chlorids and potassium determined by precipitation with platinic chlorid.

Tables 3 and 4 give the actual and the percentage composition of the stools of different types. Very loose stools contain not only a very large amount of water, but the quantity of dried matter lost daily is much greater than that lost in stools of normal consistency. It varies somewhat with the food, but principally with the amount of water.

Fat.—The fats particularly vary in most cases directly with the amount of water. A child who is losing twice as much water is usually losing twice as much fat in his stool. A very high fat loss, however, is also sometimes seen in dry, formed, soapy stools. The average loss of fat in loose stools was two and a half times that seen in normal stools. The percentage of fat varies widely; the average was 37.7 per cent. of the dried matter in the very loose stools, the highest found being 62.2 per cent. of the dried matter of the stool. The highest found in stools of normal consistency was 54 per cent.

The form of fat differs much in the different types of stools. In the normal pasty stools the soap fats are present in largest amount. The soaps form on the average 54.3 per cent., while the neutral fat forms but 39.4 per cent. of the total fat of the stool. In very loose stools these ratios are reversed. The greatest proportion of the fat (64.2 per cent.) is neutral fat, while soaps form but a small proportion (8.3 per cent.) of the total. In eight observations on infants with loose or very loose stools, no soaps at all were formed. This is what might

TABLE 4.—Percentage Composition of Dried Matter

	Stools of Normal Consistency			Loose			Very Loose		
	Seven Patients Eleven Periods			Eight Patients Fourteen Periods			Six Patients Ten Periods		
	Average, %		Range, %	Average, %		Range, %	Average, %		Range, %
Fat	35.7	.....	(10.4- 81.0)	31.2	.....	(3.3- 56.7)	37.7	.....	(6.6- 61.3)
Neutral fat	.....	34.9	(12.2- 66.7)	.....	70.4	(10.6-160.)	.....	64.2	(17.2-103.)
Free-fatty acids	.....	10.8	(0. - 65.3)	.....	17.7	(0. - 35.6)	.....	27.5	(0. - 91.4)
Soap fats	.....	54.3	(14.7-173.)	.....	12.3	(0. - 47.2)	.....	8.3	(0. - 27.2)
Protein	25.8	.....	(15. - 36.5)	27.5	.....	(18.7- 44.4)	26.3	.....	(17.9- 50.6)
Total ash	24.8	.....	(13.8- 40.9)	21.0	.....	(11.3- 45.7)	18.7	.....	(14.3- 22.6)
CaO	.....	47.0	.....	.....	42.3	.....	.....	27.4	.....
MgO	.....	4.4	.....	.....	4.0	.....	.....	4.0	.....
P_2O_5	.....	29.1	.....	.....	27.3	.....	.....	19.7	.....
Cl	.....	1.0	.....	.....	5.6	.....	.....	9.2	.....
K_2O	.....	8.3	.....	.....	14.2	.....	.....	27.6	.....
Na_2O	.....	1.9	.....	.....	6.6	.....	.....	11.9	.....

be expected to occur when the intestinal contents are hurried through the bowel too rapidly for saponification to take place. Free fatty acids represent those not combined with bases to form soaps; their amounts also were higher in the loose than in the normal stools and much higher in the very loose stools.

Protein.—The daily amount of protein lost in the stools calculated from the nitrogen is about two and a half times greater in very loose than in normal stools; it being in about the same ratio in the two as the fat. The percentage of the dried matter of the stool which is protein is nearly the same in the stools of the different classes.

Ash.—It is well known that the ash represents a considerable part of the dried matter of the stools, but that in normal, pasty stools it is equal to two-thirds the fat is not generally appreciated. In loose stools about the same relation of fat and ash is preserved, while in watery

TABLE 5.—PERCENTAGE OF DRIED MATTER OF STOOLS NOT ACCOUNTED FOR AS FAT, PROTEIN AND ASH

Stools	Dried Weight, Gm.	Sum of Fat, Ash and Protein, Gm.	Balance not Accounted for, Gm.	Per cent. of Dried Weight
Normal	8.75	7.55	1.20	13.7
Loose	12.95	10.32	2.63	20.3
Very loose.....	21.16	17.49	3.67	17.3

stools the proportion of fat is relatively higher. The amount of ash in some individual cases is interesting. In one normal stool it formed over 40 per cent. of the dried matter of the stool and in one loose stool, over 45 per cent.

When we study the distribution of the salts which make up the total ash it is seen that, while the percentage of the total varies, the actual amount of calcium is nearly the same in normal and loose stools, but is lowest in very loose stools; the quantity of all the other salts, notably sodium and potassium, however, is very greatly increased.

Residue of Dried Matter not Accounted For.—In going over our figures it was evident that there was a considerable percentage of the dried matter not accounted for as fat, protein and ash. This is indicated in Table 5.

This unexplained residue varies greatly; in some stools it was only 3 or 4 per cent. of the total; in others it amounted to 25 per cent.

of the dried matter of the stool examined. In a review of the literature of the past dozen years we have not found any attempt at explanation of what this unaccounted-for residue is. It is sometimes referred to as "other substances," but usually is entirely ignored.

Exactly what it is has not yet been determined by the direct method. The probabilities are that it is made up chiefly of the cellulose derived from the bodies of bacteria. Evidence of this is found in the carbon content of the dried organic matter after the extraction of the fat. As determined by combustion, the dried matter minus fat was found to contain about 43 per cent. carbon. As protein contains 52 to 54 per cent. of carbon, and carbohydrate but 44 per cent., this substance is

TABLE 6.—RATIO OF INTAKE TO EXCRETION AND PERCENTAGE OF INTAKE LOST IN STOOLS

	Normal		Loose*		Very Loose†	
	Seven Patients Eleven Periods		Eight Patients Fourteen Periods		Six Patients Ten Periods	
Fat	12.4	....	23.1	....	40.5	....
Protein	7.7	....	14.9	....	25.2	....
Total ash....	40.0	(in urine)	46.6	(in urine)	84.3	(in urine)
CaO	67.2	Trace	70.2‡	Trace	79.0	Trace
MgO	56.7	Trace	66.0	Trace	100.0	Trace
P_2O_5.....	36.9	36.7	41.4	36.0	48.3	41.7
Cl	3.7	79.0	17.7	70.2	52.9	17.0
K_2O.....	15.4	54.9	37.9§	37.9	83.7	26.6
Na_2O.....	12.3	67.5	37.0	28.2	103.0	5.7

*In six periods the sodium and potassium distribution was not determined.

†Complete distribution of ash was determined in only four periods.

‡Fourteen periods.

§Eight periods.

probably chiefly carbohydrate. This carbohydrate is certainly not derived from the food. The sugar ingested is either absorbed or undergoes fermentation in the alimentary tract. It is true that many of our children were taking starch, some of them in considerable amounts. But in only exceptional stools could the presence of starch be demonstrated by microchemical tests, and then only in minimal quantities. It was in no case sufficient to account for the carbohydrate content of the stool. Furthermore, just as great carbohydrate content existed in the stools of many children who were receiving no starch

at all. The evidence therefore all points to cellulose derived from the bodies of bacteria as the chief factor in this dried matter unaccounted for.

The proportion of the stool made up of bacteria is great, making, according to various investigators, from 10 to 40 per cent. of the dried matter; in some constipated stools in infants it is estimated to have made up two-thirds of the stool.

Ratio of Intake to Excretion.—In the normal stool the loss of fat was but 12.4 per cent. of the intake, while in the very loose stools it reached 40.5 per cent. It is known that fat may be demonstrated in the stool when none is given in the food, it being derived from the intestinal secretions; but the amount is very small and from a nutritive point of view it is a negligible quantity. The loss of protein in the normal stool estimated from the nitrogen content represents but 7.7 per cent. of the intake. This shows how completely the protein of cow's milk is digested and absorbed by the average child. In the loose stools, calculating all the nitrogen as representing protein, the loss is 14.9 per cent. of the intake, and in very loose stools it is 25.2 per cent. This is not strictly true. In loose stools, and especially in watery stools, a considerable amount of protein present is not derived from the food, but is drained away from the body in the intestinal secretions. How much this is we are as yet unable to estimate, but it must be quite large. It is probable that although the infant with diarrhea loses over 40 per cent. of the fat which is given, he does not lose 25 per cent. of the protein given. This is incidental, but very important confirmatory evidence of the value of protein feeding in diarrhea which has received so much support from clinical observations. When we study the distribution of the salts which make up the ash it is evident that they are not derived entirely from the food ingested, but that they represent a considerable draining away of salts from the body itself. In normal stools nearly four-fifths (76.1 per cent.) of the ash is calcium oxid and phosphoric acid, probably existing in the stool as calcium phosphate, while the proportion of chlorin and potassium and sodium oxids together form but 11.2 per cent. of the total. In the dry, formed stools the ash is often high, due to the bases combined with the fats as soaps.

In very loose stools we found calcium oxid and phosphoric acid to form but 47.1 per cent. of the total, but there was a great increase in the proportion of sodium, of potassium and of chlorin; together they

make up 48.7 per cent. of the total ash, the chlorin being nine times, the potassium oxid three and a half times, and the sodium oxid six times the proportion in normal stools. This cannot be without significance.

Normally, 40 per cent. of the ash intake is lost in the stools; this is chiefly calcium phosphate, of which cow's milk has so great an excess. In very loose diarrheal stools there is lost 84.3 per cent. of the intake. The proportion of calcium phosphate shows but a moderate increase over that in normal stools, 79 as compared with 67.2 per cent. of the intake. The great increase in the total is made up of the other salts. The magnesium loss is 100 per cent., indicating that it very slowly enters into combinations in which it can be absorbed. The loss of calcium and magnesium occurs almost exclusively in the stools, only traces being found in the urine. Of the remaining elements, the loss in the urine must be considered in estimating the balances. Chlorin, potassium and sodium are normally present in the stools in relatively small amounts, but in the urine in large amounts. In diarrheal stools we find an enormous increase in the quantity present in the stools and a corresponding reduction in the amount eliminated by the urine. But the increase in the stools is so great as to create a negative balance of 10.3 per cent. in potassium and 8.7 per cent. in sodium. This indicates the loss from the body itself.

The ratio of the loss of salts to the intake is one of the most striking characteristics of loose, diarrheal stools and furnishes an important suggestion as to treatment. In attempting to supply this loss by hypodermoclysis it should be remembered that not only are water and sodium needed, but potassium and magnesium as well. With these facts in mind a better solution for use in hypodermoclysis can certainly be devised than a normal saline or than Ringer's solution.

This subject is still under investigation at the Hospital Laboratories and the foregoing is to be regarded in the nature of a preliminary report.

DISCUSSION

DR. SOUTHWORTH: This is extremely interesting work which Dr. Holt has begun to do, and pioneer work, I think, as he has stated. I would prophesy that a good deal will come from these studies of the question of the loose stool and the formed stool, and that more will eventually be learned in the way of improving the nutrition of the child by methods which are directed particularly to bringing the condition of the intestinal digestion to such a point that the stool will pass more slowly, and in more solid form, through the intestine. Some work has just been published in the *Jahrbuch für Kinderheilkunde* on this subject. Just how this will always be possible I do not yet know; so much

depends, in the infant's nutrition, on the simple question of whether the stools are hurried through the intestinal tract, without opportunity for absorption, or whether the digestion of the food is such as to split it into irritant bodies, which cause an increased peristalsis and escape of the intestinal contents before proper absorption can take place.

When Dr. Holt closes, I should like to ask him whether he has met the paradoxical condition that a constipated child often loses weight during the twenty-four hours during which the constipation persists, and whether he has any theory for this paradox. One would, of course, naturally suppose that with the retention of the solid matter there would tend to be an increase of weight, but I think those of us who have followed the daily weights of children in the hospitals have often noticed that a short period of constipation is accompanied by a loss of weight.

DR. TALBOT: I have been very much interested in Dr. Holt's paper. He said that they had no way of telling about the carbohydrate. We have been estimating the splitting of carbohydrate in a very crude manner, but I think that it has given some suggestive results, namely, by quantitative acidity of the stools. I say it is very crude, because the titration of acidity against any indicator is crude. When the digestion of carbohydrate gets beyond a certain point there is a marked increase in the acidity of the stool. In one instance it increased from 50 c.c. tenth normal sodium hydrate to 250 c.c. of tenth normal sodium hydrate. At present, we are studying the acidity of the stools by means of the hydrogen ion concentration of the stools, and hope that that will throw further light on the subject.

DR. KERLEY: If retention of chlorids is a factor in the production of tetany, it would seem that diarrhea would be productive of the relief of the condition. I think it would be very interesting to have Dr. Holt speak of that feature of his study when he closes.

DR. HOLT: As I indicated at the ouset, we have only made a beginning in this study. It has seemed to me that if carried on for a longer period and by many observers, we might get some really valuable assistance in the feeding of diarrheal patients. Up to the present time almost all the work done on the diarrheal stools has been bacteriologic.

I can not answer Dr. Southworth's question as to why it is that a child with a large mass of constipated feces in his bowel weighs less than a child with the feces removed.

In reply to Dr. Talbot, I may say we have done some work on the acidity of the stools and found that constipated stools are frequently alkaline, and the loose stools usually strongly acid.

Replying to Dr. Kerley's question, we made the observation more than a year and a half ago that if a child, in the course of tetany, for any reason developed an acute diarrhea, his tetany was promptly relieved, or, at least, was much improved. The characteristic electrical reactions from being very marked would nearly or quite disappear, sometimes in the course of from twelve to twenty-four hours. The next step was to induce the diarrhea by producing a free, active catharsis, when we found that the same result followed. The great amount of sodium and potassium noted in the examination of these loose stools suggest that the elimination of these substances might have had some influence on the nervous irritability of these patients.

ONE HUNDRED AND FORTY-ONE CASES OF RECURRENT VOMITING IN PRIVATE PRACTICE

CHARLES GILMORE KERLEY, M.D.
NEW YORK

The cases reported have been seen in private practice during the past eight years. Cases seen in consultation with other physicians have not been included.

The history diagnosis was based on definite periodic attacks of vomiting, for which condition the patient was brought to me.

Recurrent vomiting, in a vast majority of the cases, we found occurs in the offspring of those who for two or more generations have not been occupied with manual work. The recurrent vomiting patients are the offspring of those who have had business or professional occupation.

SUMMARY OF THE FEATURES OF CASES SEEN

Average time cases were followed was 104 weeks.

Sex: Seventy were boys; seventy-one were girls.

Family History: Rheumatism in one or both parents in forty; sick headache and bilious attacks in forty.

Appetite: In 31 per cent. was normal; in 69 per cent. it was indifferent; in 59.5 per cent. there was constipation.

Previous Feeding in 120 Cases: Twenty-one patients were nursed by mother nine months; forty were nursed by mother six months; four cases had been wet-nursed; sixty-nine had been difficult feeders on cow's milk and had been given the usual trial of foods.

Recurrent colds had been present in 41 per cent. This information was usually volunteered. In twelve there was definite history of eczema; six had habit tic. In fourteen there had been or was enuresis. In thirteen there had been rheumatism.

Urine: Nothing was known as to the condition of the urine in the attacks before coming under observation. The presence of acetone in the later attacks was noted with very few exceptions, but there were exceptions. A child in an attack may show acetone and in the next attack the urine may be free.

Onset of the Vomiting Seizures: Thirty-seven occurred during the first year; twenty-four occurred during the second year; twenty-one occurred during the third year. Onset in the youngest was 6 months. In three the onset was during the eighth year.

Duration of Interval Between Attacks in 119 Cases: Under 2 weeks, 22; from 2 to 4 weeks, 27; from 4 to 6 weeks, 10; from 6 to 12 weeks, 42; from 3 to 6 months, 40.

Duration and Severity of Seizures: The severity of the attacks varied widely; in some the child would vomit but two or three times. These usually represented the short-interval cases. In others the vomiting was protracted and severe. The average was three and one-half days. The longest seizure under observation continued thirteen days.

The average interval in 118 cases was eight and one-fourth weeks.

Temperature: 102 F. or over in 30; 103 F. or over in 10; 104 F. or over in 6; 105 F. or over in 3. In the remainder there was an elevation of from 100 to 102 F.

In taking the history of fifteen cases, the mother or attendants volunteered the information that the child had a peculiar breath at the time of the attack which we interpreted as the so-called "acetone breath."

Onset: Average age in 121 cases was 2 years and 8 months; average number of attacks in 99 cases had been 9; average age brought for treatment was 5 years.

Results: Of cases in which there were no attacks after beginning treatment: Forty-one were followed for from two to eight years. Of these forty-one, twenty-six were followed from three to eight years and fifteen were followed from two to three years. Fifteen were followed from one to two years; nine cases were followed from six months to one year; eighteen cases were followed less than six months. Several of the latter are recent cases in which decided gain in weight and general improvement has occurred. In nine cases there was very much improvement; the attacks were mild and comparatively infrequent. Of these eight were followed for from two and one-half to eight years; one was followed eighteen months. In thirty-three cases there was improvement; the attacks less frequent and less severe. Of these thirteen were followed from two and one-half to eight years; six were followed from one year to eighteen months. The remaining fourteen of this group were followed for varying periods under one year, but the records show that a decided impression had been made on the seizures.

In sixteen cases there was no apparent improvement. In some the treatment was indifferently carried out and the patient was dismissed. In others, no apparent impression was made on the disease and the seizures continued as before.

I have an unimproved case that has been under treatment for three years without benefit. The attacks continue to be very severe at from ten to twelve weeks' interval. I only recently was successful in having massage and exercise treatment instituted.

Another boy now aged 6 has attacks of either periodic fever, bronchitis or recurrent vomiting, rarely going for three months without an explosion of some kind. He has been my patient since he was 6 months of age.

In a vast majority of the cases the recurrence will be completely controlled if continued family cooperation is secured. A diet and living regimen will be followed faithfully by some parents as long as they can be kept anxious about the patient. When the child has passed

three or four vomiting periods, the use of forbidden articles of food is often gradually resumed; the former methods of living are more attractive than the regimen laid down.

The association of recurrent vomiting with other forms of illness is interesting. As mentioned before, 41 per cent. of these children were subject to repeated bronchitis, usually of the spasmodic type, and not infrequently the bronchitis and the vomiting occurred simultaneously. Acetone was not present in these cases unless gastric symptoms were present. In others, the attacks will be distinct and separate. In one patient there was in nearly every attack an acute spasmodic laryngitis; bronchial asthma very urgent followed and this again was followed sometimes, but not invariably, by repeated vomiting. This child had suffered severely with eczema when an infant. Two boys, brothers, were of particular interest in that they had the attacks simultaneously, beginning with tonsillitis, followed by bronchitis and asthma and ending with recurrent vomiting lasting for a day or two. In a few cases the recurrent vomiting attacks were replaced by high fever for three or four days, with acetonuria and without other symptoms.

Two girls, aged 5 years, both in wretched condition, had mild vomiting every week or ten days. In both the acetone breath and acetone urine were continuously present.

The only fatal case seen by me and not included in the above statistics was in a girl 7 years of age living in a New York suburb. There had been previous attacks at about from three to six months' intervals. This attack was particularly severe, no fever but frequent and violent emesis. The vomiting had continued about four days when I saw her. At this time there was considerable exhaustion, but the child was fairly bright, answered questions and talked freely. I endorsed the attending physician's treatment, made a favorable prognosis, and the child died in five hours. Apparently, death was due to respiratory paralysis.

Three patients had been operated on for appendicitis in Paris without relief. A normal appendix was removed in each case. In three it was necessary to resort to morphin hypodermatically to control the vomiting seizures.

MANAGEMENT DURING THE INTERVAL

The management in the main was the same in all, and one reason for reporting only the cases seen during the past eight years is that during this period practically the same interval management has been carried out.

Diet: If the case is a pronounced one, the patient is given a diet with few restrictions, except that cow's milk, butter, cream and sugar are omitted. One egg is allowed perhaps every third day. Saccharin is permitted as a sweetening agent in some and very little sugar in any case.

Three meals daily are allowed with nothing between meals. Red meat is given scantily three times a week. Poultry and fish are given at other times. In some skimmed milk is allowed scantily, never more than one pint daily, often less. Puddings are made with skimmed milk. A grave error in our management of many children is the free use of cow's milk, butter, ice-cream and sugar. I could present dozens of records showing surprising gain in weight and marked improvement in the general well-being of the patient after a considerable withdrawal of milk, cream and sugar from the diet.

The period of lactation in the human being is at the most a year and then the child is ready for other food than milk. I believe that the average well child would thrive far better if he were to get not more than one pint of milk daily after the fifteenth month. Sugar was not used except as a condiment as we now use honey, until three hundred years ago. Unknown millions lived their span without it.

Medication: The further treatment consists in the internal use of salicylate of soda, bicarbonate of soda, independent or in combination as advocated by Rachford. In a pronounced case I give 5 grains of sodium salicylate with 10 grains of sodium bicarbonate three times daily at five-day intervals, or 20 to 30 grains of the sodium bicarbonate daily for a month or two at first. This drug treatment is carried on with rest periods for months and years as the case may require.

Bodily Exercise: A very important factor in the management is in arranging daily physical exercise, such as riding horseback or the bicycle and walking so many street blocks a day. A warm bath and a brisk rub are given at night, and last but not the least important feature in the treatment is the use of physical therapeutics. In severe cases I use if possible daily massage together with various body manipulations and exercises, the latter sufficient to make the child perspire, but not to the point of exhaustion. The advantage of this phase of the treatment was called to my attention by Dr. W. P. Northrup.

Bowel Function: A daily evacuation of the bowels is insured by suitable measures.

Some children I take out of school. For others I advise a modified rest-cure, which means in bed until 10 a. m., rest one and one-half hours after dinner and in bed and lights out at 6 or 7 p. m. The nervous element in these cases is not to be forgotten. If the attendant is not agreeable to the patient her services are dispensed with. In some a temporary elimination of the mother has been of assistance. The precipitation of an attack by fatigue and fright is not unusual.

The regulation of the life and habits aids materially in the management, but is of little or no use if the carbon content in the food is not reduced to the oxidizing possibilities.

MANAGEMENT DURING THE ACUTE VOMITING ATTACK

I find that in the vast majority of the cases, a weak solution of sodium bicarbonate is best retained in the strength of 5 grains of the bicarbonate to 8 ounces hot water. This is given freely.

As laxatives the magnesia preparations are best retained and are used when a laxative is required. Calomel or mercury with chalk will often increase the vomiting. In other cases their use supplies very material assistance. When the vomiting has continued for twenty-four to thirty-six hours the patient is given colonic flushings with sodium bicarbonate, 2 drams to 8 ounces of water at eight-hour intervals. The solution is best retained if it is given warm (105 F.), the tube inserted from 8 to 12 inches.

Feeding: Nothing is gained by attempts at forcing the feeding. When the child is ready for food, he is given barley or rice gruel with dried bread crusts or unsweetened zweiback.

Judging from the results obtained through the withdrawal of highly energized foods and in the use of active and passive exercises it would seem that the chief error in most cases rests in a defective oxidation, or in the giving of food substances of high carbon content in excess beyond the powers of normal oxidation.

In this connection the views of Francis Hare[1] are particularly interesting. Hare states that carbon intake must be offset by carbon expenditure or energy intake with compensating energy expenditure, if "hyperpyremia" (excess of fuel in the blood) is not to occur. When physiologic functions are deficient in maintaining a balance, and an excess of carbon or hydrocarbon is not wholly applied in the manufacture of additional fat, bile, milk or other secretion, or lost by exercise

1. Hare, Francis: Carbon Factor in Gout, Med. Rec., June 17, 1905.

or menstruation, a pathologic function is then necessary to free the system of its excess of fuel. As such so-called pathologic functions, Hare mentions gout, migraine, gastralgia, bilious attacks, epilepsy and asthma, all of which operate through either a reduction of income or an increase of expenditure by vomiting, muscular action or fever, periodic in character. These attacks of migraine have been observed alternating in the same individual with attacks of gout; gout has subsided with the development of glycosuria, and women (according to Garrod) show greatest liability to the development of gout shortly after the menopause, one decarbonizing process replacing another.

I am not convinced of the reliability of this theory in its entirety, but find it peculiarly in harmony with the clinical facts under presentation, and (while not forgetting for a moment that proteid anaphylaxis (Meltzer) and reflex neuroses from various abnormalities are still to be considered as operative causes in such affections as asthma, recurrent vomiting and migraine) I wish to emphasize the following suggestive points:

Eczema, spasmodic laryngitis, cyclic vomiting and recurrent bronchitis and asthma are all notoriously frequent in children of gouty or rheumatic ancestry.

All these conditions are met less frequently and with diminishing severity at the age of puberty when the processes of combustion and tissue building are at their maximum.

While two or more of these so-called pathologic functions are not ordinarily observed simultaneously in the same child, not a few children suffer from a number of these conditions in alternation over a period of years. In other words, the processes seem to be mutually compensatory.

In the winter months, when activity is lessened and perspiration is least, every one of the conditions mentioned is intensified.

Finally, in every instance, diet (as the cases show) is a most prominent factor influencing the susceptibility of the patient.

The statistics as presented were prepared from my case histories by my associate, Dr. Frank Elmer Johnson, to whom I wish to express my indebtedness.

132 West Eighty-First Street.

DISCUSSION

DR. SNOW: One of the first patients I ever treated suffered from recurrent vomiting. The child was of alcoholic ancestry; suffered convulsions in infancy; was a perfect type of the spasmophile diathesis. She had recurrent attacks of vomiting, very severe but not periodic. The vomiting usually lasted three

or four days. The ejecta was a highly acid gastric juice, the free hydrochloric acid amounting to from 30 to 40 points. Evidently there was a frequent hypersecretion of gastric juice which irritated the pylorus and caused distressing vomiting.

No examination of a patient with recurrent vomiting is complete without a chemical test of the fluid vomited.

Last year I treated a child in which there was reason to believe that the vomiting was caused by a duodenal ulcer. During its infancy it vomited a portion of every meal. At two years the child commenced to have severe vomiting attacks every six or eight weeks. As time went on these illnesses recurred at longer intervals and grew more severe.

By her sixth year the vomiting produced such exhaustion that several times the child seemed on the point of death. The child would be practically moribund; would be given a hypodermic of morphin and recover again. Large quantities of blood were vomited, mixed with gastric juice containing from 40 to 50 points of free hydrochloric acid. A consultant, Dr. Woehnert, considering the hemorrhage and hyperchlorhydria, suggested the possibility of a duodenal ulcer, and discovered a dilated stomach.

A diet appropriate to duodenal ulcer—fats, cream, butter, bacon—with the idea of reducing the acidity of the gastric juice, was given, also alkalies after meals. A rapid improvement occurred and no severe attack has occurred in eleven months.

This case of recurrent vomiting was treated on the theory of a peptic ulcer.

The point in the discussion I would like to make is that the vomited fluid should be examined for free hydrochloric acid for diagnosis and also for treatment, and that a certain proportion of these cases of recurring hyperchlorhydria are due to duodenal ulcers and are enormously improved by a diet containing fats—an idea at variance with Dr. Kerley's treatment. The examination of urine is a waste of time.

DR. DUNN: I am much interested in this subject, because I have been collecting a similar series of cases (not so large as Dr. Kerley's) which have occurred in my practice, and which I have seen in association with Dr. Rotch; and I am particularly interested in the possible relation of these cases to acid intoxication. The patients in this series of cases have, as a characteristic symptom complex, the association of sudden and persistent vomiting, without known dietary cause, with the presence of the acetone bodies in the urine. My cases seem to be divisible into two types. One type begins like an attack of recurrent vomiting, but the picture soon changes, and the vomiting remits or ceases; in the meantime the patient does not get better, but gets worse, and a marked nervous intoxication comes on, which may be fatal. This type of case begins like those described as recurrent vomiting, but it does not appear to be recurrent, and it is influenced, favorably, I think, by the administration of large doses of sodium bicarbonate. It seems to me there is evidence of good clinical results from that treatment, and therefore they seemed to me to be true cases of acid intoxication. The other type of case has no nervous system intoxication, and vomiting is the main symptom throughout. These are the typical cases of recurrent vomiting such as Dr. Kerley describes, in which there is a tendency to recurrence. I have tried in this type the same treatment, with large doses of sodium bicarbonate over and over again, both in the interval and in the attack, and I have never been able to convince myself that there was the slightest evidence of any therapeutic benefit from the giving of sodium carbonate in these cases.

I have realized that the probable opinion of the majority of this society would be against me on that; but that, in fact, has been my observation, and now I have even taken to giving up the use of sodium bicarbonate. I have seen two fatal cases of recurrent vomiting, in both of which death occurred from gastric hemorrhage, and they were cases which were persistently fed by mouth in spite of the vomiting. In them the vomitus gradually came to contain more and more blood, and they made such an impression on me that I have come to the conclusion that hemorrhage is the chief danger in the true type of recurrent vomiting, and that the essential of the treatment is rectal feeding from the beginning of the attack. We used to give the sodium bicarbonate partly by rectum, but now I do not want to run any risk of the rectum becoming intolerant for any such doubtful benefit as sodium bicarbonate confers. I believe that in this type the acetone bodies are merely symptomatic and not causative. The first type, on the other hand, which is not recurrent, and often fatal from intoxication, I believe to be a true acidosis.

DR. FRIEDLANDER: I desire to make a preliminary report of a study which Professor Fischer and I are engaged in with reference to recurrent vomiting. The failure of the alkali treatment in certain severe cases has led us to the belief that it might be worth while to try out certain glucose solutions in the hope that they might be more effective; and we have now been using for some time a solution of dextrose and sodium chlorid: 20 gm. of dextrose and 14 gm. of sodium chlorid to the liter. While it is not possible to make an exhaustive report, the results so far, I think, have been surprisingly good. The attacks have been cut short very rapidly; and the profound exhaustion following the very acute attacks has not appeared. The children have rallied from the attacks much more promptly than they have from the previous attacks during the treatment by the older method. So far as the recurrence is concerned, we have had the cases under observation too short a time to be willing to make any definite report; but I desire to place this communication on record at this time because of the excellent results following its use.

DR. ABT: I wish to corroborate the remarks of the last speaker from the experience I have had in the treatment of these cases. First I wish to say that I do not believe that the diet during the interval has any effect on the recurrence of the attack. They will occur just about as frequently whether we feed them this, that, or the other; and it does not seem to make any particular difference as to their exercise or their general mode of life except such things as influence their nervousness. Whatever increases nervousness is likely to bring on an attack; an acute infection will occasionally bring on an attack.

I wish to corroborate what Dr. Friedlander has just said. For a long time we have been treating these cases, by the advice of a chemist in Chicago, with 8 per cent. glucose solutions in normal salt solution. Just as soon as possible we start by giving him toast, or cracker, or zwieback, or gruel, and we find the attacks are very much diminished in severity and lessened in duration.

Then, too, we give sodium bicarbonate in all these cases, and personally I think I have seen results by giving them frequently ice-cold chloroform-water.

DR. GRIFFITH: What I have heard to-day has convinced me of the truth of the conclusion to which I came when I reported some cases of this disease a number of years ago, namely, that we do not know just what the cause of recurrent vomiting may be. There are cases which can, apparently without doubt, be benefited by the administration of sodium bicarbonate, but it is

equally certain that there are others not at all benefited by it. I have seen instances of this sort, even when sodium bicarbonate in large doses was continuously administered between the attacks.

Regarding the title "recurrent," I would urge that this is a much better term than the word "cyclic," often employed. The latter would designate a certain degree of regularity or periodicity, so to speak, and this does not exist. "Recurrent vomiting" has no such signification. It is certainly my experience that many cases are benefited by the alteration of diet, as, perhaps, by the withdrawal of all fat from the food; but there are certainly other cases, so far as my experience has gone, in which nothing that I could do in this way of diet had any influence in preventing the attacks. The majority of instances which occur are of comparatively mild nature. They might be called of trivial importance as compared with the more severe ones. These severer cases are truly dreadful instances of vomiting. The child finally becomes so exhausted that it lies on its side, and now and then a small stream of colored mucus runs from its mouth, weakness being so great that violent vomiting is no longer possible. In those cases in which death seems to be impending and you feel that something *must* be done, I have found nothing so serviceable as one or more hypodermic injections of morphin. Elimination has probably completely taken place, and you are now dealing with what might be called the persistence of the habit of vomiting, and morphin seems to be the best remedy to control this. I have seen two fatal cases, another patient that I believe certainly would have died if morphin had not been given, and another in whom weakness became so extreme before the remedy was employed that thrombosis occurred in the vessels of the leg, and gangrene, with subsequent loss of the foot, took place. Many of these cases simulate strongly intestinal obstruction in the presence of the obstinate constipation and vomiting; and the diagnosis is often difficult and uncertain.

DR. HEIMAN: We all know under how many names this condition is described in the text-books or literature: cyclic vomiting, recurrent vomiting, acidosis, auto-intoxication, acetonuria or pancreatic insufficiency. I believe those cases clinically are mild, moderate, or severe. The real causative factors are unknown at present, but we believe there is a metabolic disturbance. The acetone itself is not eliminated as such in the urine, but is only formed from diacetic or beta-oxybutyric acid by the addition of chemicals. We also know that acetone may be present in any of the infectious diseases. If a child has a tendency to this disease, the very first thing I order is sodium bicarbonate. Some of these cases may end fatally, not from the acidosis, but from complications. In one instance a patient of mine developed eclampsia, with a temperature of 108 F., and died very promptly. I do not think any treatment would have saved the child. The mild cases respond readily to the internal administration of sodium bicarbonate in peppermint water. I much prefer using peppermint water, because of its cooling analgesic effect. I give sodium bicarbonate by mouth in very small amounts; by rectal administration, we will say ½ ounce to 1 ounce of water with 15 grains of sodium bicarbonate and, if necessary, by hypodermoclysis, or even intravenous injections. I also order in these cases intermittent alkaline treatment, which acts as a prophylactic measure.

DR. RUHRÄH: I have been much interested in these cases for many years, and have seen many. We are placing and trying to group under one clinical head five or six different things, and the result is considerable confusion. I think you can group them satisfactorily, from a clinical point of view, some-

thing like this: First, we have certain attacks of vomiting which may recur at intermittent times, which are really the action of food anaphylaxis, food idiosyncrasies; secondly, we have these acid intoxications, which sometimes come from errors of diet and at other times are apparently caused by other things—such as the administration of anesthetics, administration of salicylates, sometimes from conditions we do not understand; and, thirdly, we have recurring attacks of vomiting from food poisoning. We can have these attacks I believe, from excess of fats, from excess of proteins or from excess of carbohydrates. I am sure I have seen cases which fell under all three of these groups, and in which the cases have been relieved for years, with only an occasional attack, by simply cutting down the offending substance; and, lastly, one sees—I think quite often—recurring attacks of vomiting at more or less irregular intervals, in which the vomiting seems to be the effect of some reflex nervous condition. In those cases it is very difficult to say just what the process is, but we see these cases and the vomiting recurs at more or less irregular intervals.

DR. SHAW: I think a little more emphasis should be placed on the fact that some of these cases are fatal. I had the same unfortunate experience that Dr. Kerley had in giving a most positive favorable prognosis in a case of this character. This was a child from Baltimore and had been under the care of some of the best Baltimore men and was a typical case of recurrent vomiting. I saw the child Sunday morning, the third day of its attack: it was pretty sick. The acetone was very evident in the atmosphere of the room and the urine contained acetone and diacetic acid. We started giving even larger doses of sodium bicarbonate than had been given by the nurse but about 8 p. m. the child died.

DR. KNOX: With reference to the use of atropin, I think there are a certain number of these cases, especially when there is hyperacidity in older children, in which the use of atropin pushed to its physiologic limit until the child flushes, in connection with starvation, and the use of sodium bicarbonate, and sugar, perhaps by the bowel, is of very great advantage.

DR. NORTHRUP: The case referred to by Dr. Kerley we saw together. The case was under treatment, very skilful handling, for a couple of years; what it finally yielded to, I think, was, in both of our minds, massage. I had a particularly skilful masseuse, who was very devoted, very agreeable to the family, very tactful with the child; and she certainly, in some way, although it was a very obstinate case, brought, I think, complete recovery. I would like also to add that she was a very valuable nurse, very valuable to me; and after Dr. Kerley got hold of her I never saw her again for two years; so I want to get square with him at this moment.

DR. KERLEY: I have no theories to offer. My paper is an analysis of a given number of cases taken from my case records. In recurrent vomiting there appears to be a defective oxidation and the best results are obtained regardless of theories when highly energized foods are removed from the diet and when means are used to increase the oxidizing capacity of the organism.

WEIGHTS AND MEASUREMENTS OF INFANTS AND CHILDREN IN PRIVATE PRACTICE COMPARED WITH INSTITUTION CHILDREN AND SCHOOLCHILDREN

ROWLAND G. FREEMAN, M.D.
NEW YORK

Information as to the weights and measurements of children is only beginning to be reliable. Much that is erroneous has been published concerning the weights during the first year and very little data has been published as to the gain in weight and height of children under the good conditions that usually prevail in the feeding practice of pediatricians.

While believing that standard weight charts are an absolute disadvantage when given to the family, there can be no question but that a physician feeding a baby should have certain standards in mind, although each baby will supply a standard of its own.

While most of us have the care of a considerable number of children under very good conditions where the feeding can be, at least during the first years, absolutely controlled, where the exposure to fresh air, the amount of exercise, the amount of rest and the character of the clothing are all supervised, we have had little definite knowledge as to whether the results obtained for these children were commensurate with the care that was given them. We know in a general way that the mortality of these children is much less than that of children in general. For while the mortality in New York during the first year, which was, some years ago, 30 per cent., is now about 10 per cent., I believe the mortality for well-cared-for babies during that period to be not more than one-half of 1 per cent. Of this fact again we have no definite data, but of the 120 consecutive cases in private practice that I have tabulated, not one died during the first year.

There has been accumulated now a considerable number of weights and measurements of schoolchildren in the United States, while the only data that I know of the weights and measurements of children in private practice are those published by W. Camerer.[1] It therefore

1. Camerer, W.: Jahrb. f. Kinderh., 1901, lii, 381.

seemed of interest to me to make an average weight chart of 278 cases in private practice, as well as a chart showing their measurements, and in order to get definite data for comparison I weighed and measured the 1,000 children, about 500 girls and 500 boys, in the Roman Catholic Orphan Asylums of New York. These institutions are now situated on a high bluff overlooking the Harlem River, with modern, sanitary buildings, well lighted and with ample grounds, so that while these children are institution children they are really under good conditions, and there was almost no sickness among them at the time I made these observations.

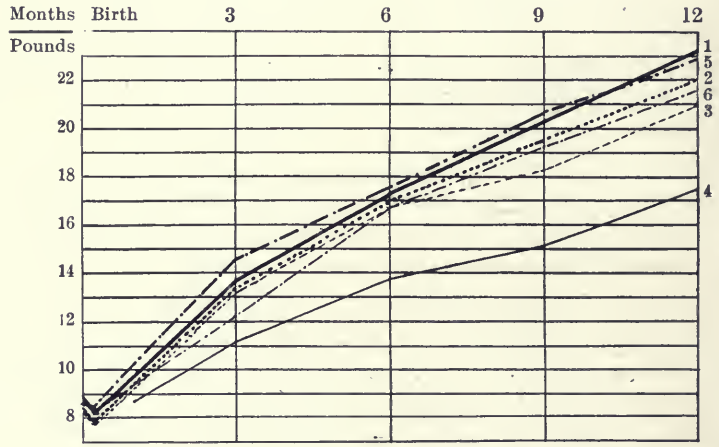

Chart 1.—Weight during the first year of 120 well-cared-for children ——— (1), compared with the average given by Dr. Holt ═══ (2 and 3) that of 500 institution children ——— (4); that given by Dr. Cammerer for 119 breast-fed children — — — (5), and 84 artificially fed children — - — - — - — (6) in private practice.

I have prepared several charts in which I have compared the weights and measurements of the children in private practice, the orphan asylum children, and such data as I have obtained from other observations.

Chart 1 shows in the heavy black line the weights of 120 well-cared-for children, consecutive cases in my private practice, while the light line shows in contrast the average weight during the first year of 500 institution children.[2] It is noticed that these lines are well

2. Fleischner, E. C.: Arch. Pediat., 1906, xxii, 740.

separated after the first month. The dotted lines which run well below the well-cared-for children, are taken from the different editions of Dr. Holt's book.[3] The lower line is the one used in the earlier edition (Second Edition, 1902) and "is made up from complete weight charts of 100 healthy nursing infants who were thriving and weighed every week, and the incomplete charts of about 300 other infants"; while the upper one is the one used in the last edition (Sixth Edition, 1911) and is made up from observations on the same number of children as the lower line. Both of these lines are seen to be below the well-cared-for children. The two dot and dash lines are the weights published by Camerer, the upper line representing breast-fed children

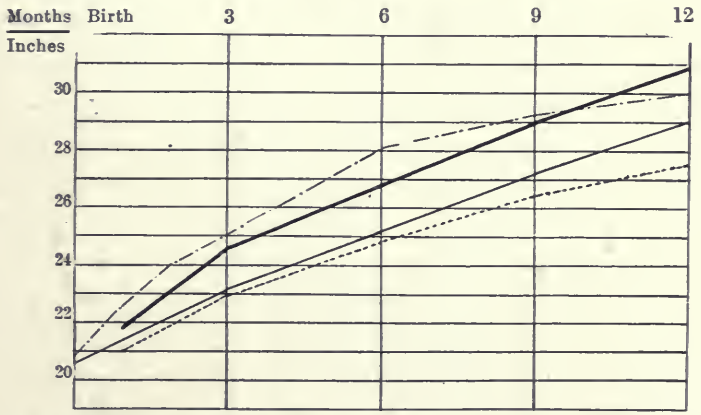

Chart 2.—Height during the first year of 120 well-cared-for children ——— compared with the average given by Dr. Holt ———, that of 500 institution children - - - - and that of 34 children given by Cammerer — — - - —.

and the lower line the artificially fed children. The breast-fed children show greater weight throughout the first nine months, while the artificially fed children keep fairly below. I may add that the 120 well-cared-for children represented in the chart were almost entirely artificially fed after the first month, so that the American method of feeding children during the first year would seem to compare favorably with the method advocated in Germany.

Passing to Chart 2, we have in the heavy black line, again, the length of the same 120 well-cared-for babies that are represented in the other chart, while the lower, dotted line represents the measurements of the

3. Holt, L. E.: Diseases of Infancy and Childhood.

500 institution children, and the light black line is the line taken from Dr. Holt's book, while the dot and dash line represents the measurements of thirty-four children reported by Camerer with no information as to whether they are breast fed or artificially fed. His line, however, corresponds to that of the breast-fed babies in the weight chart, being

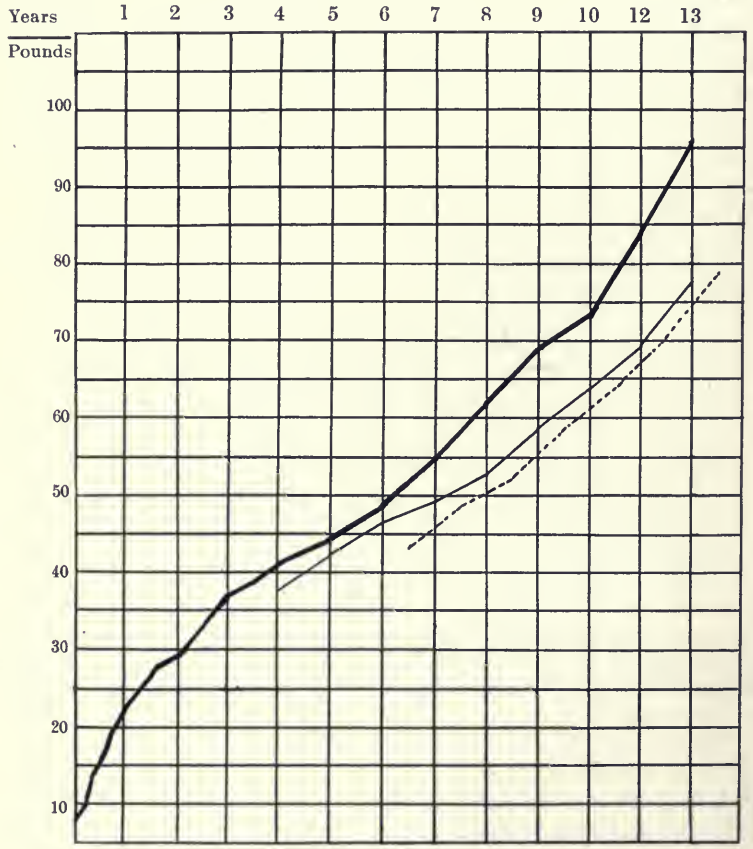

Chart 3.—Weight in pounds of 278 well-cared-for children ——— compared with 1,000 orphan asylum children ——— and 69,000 schoolchildren - - - - - -.

above my line at the first nine months and ending below it. It must, however, be considered that the thirty-four cases reported by Camerer are an insufficient number for the formation of conclusions, as there are only one to five observations on any given date.

The observations on children after the first year are represented in Charts 3 and 4.

Chart 3 shows in the heavy black line the weight of 278 children in private practice as compared with the 1,000 orphan asylum children in the light black line, and with 69,000 schoolchildren in the dotted line.

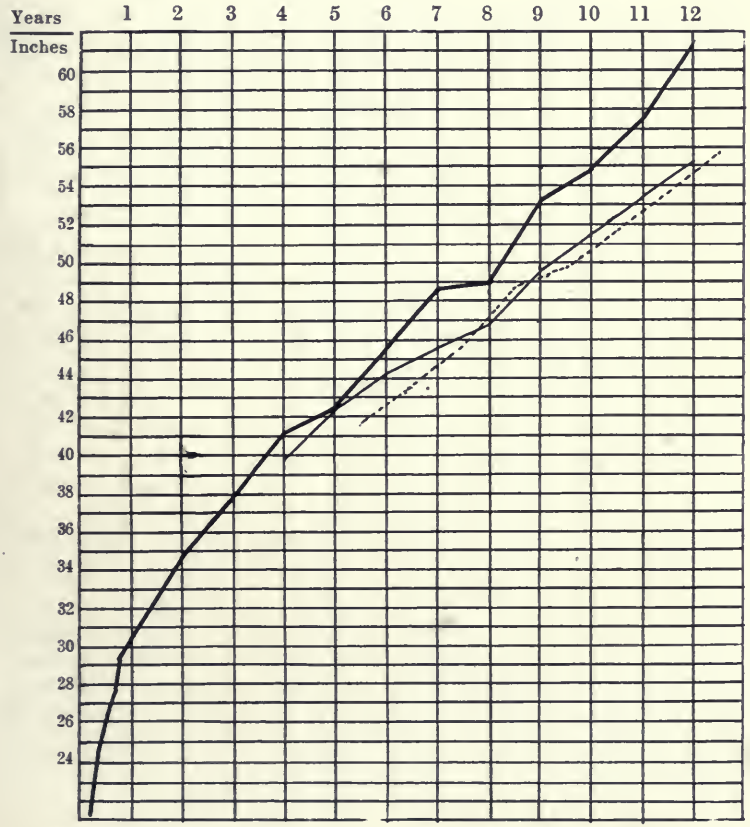

Chart 4.—Height in inches of 278 well-cared-for children ——— compared with 1,000 orphan asylum children ——— and 98,000 schoolchildren - - - - - -.

of various cities of the United States, as reported by Stanley Hall. It is noticeable that while from the fourth to the sixth year the orphan asylum children average two or three pounds less than mine, these lines become farther apart with advancing age. It is an evidence of the excellent care given these orphan asylum children that they average

2 or 3 pounds heavier than the 69,000 schoolchildren. Both of these groups, however, from 6 years on, are well below the well-cared-for after several visits from a social worker, the child was not brought cared-for children.

The last, Chart 4, which represents the length of the 278 well-cared-for children in the heavy line as compared with the 1,000 orphan asylum children in the light line, and 98,000 schoolchildren of the United States as reported by Stanley Hall, in the dotted line, shows the orphan asylum children again to average somewhat taller than the schoolchildren, both lines being well below the well-cared-for children. This shows a surprising advantage of the well-cared-for children over the others at the twelfth year, of 6 inches greater height.

In conclusion, I would say that the children that are under good control so far as diet, rest and exercise are concerned, show a great advantage over the data at hand concerning other children both in weight and height during the first twelve years of life; and that at the twelfth year they surpass the average by 20 pounds in weight and 6 inches in height.

211 West Fifty-Seventh Street.

DISCUSSION

DR. SOUTHWORTH: I shall, perhaps, be repeating some of the things that I have said many times before in what I have to say about this question of the weights of children. To start with, if we take the average weight of a child at 1 year to be 21 pounds, as I think Dr. Holt gives it, or to be 19 to 20 pounds, as I think Dr. Chapin gives it (and these latter figures by the way, always seemed to me to be somewhat low for the normal child in private practice), we mean by that that a large number of the children have reached 1 year of age considerably below weight; because we know that a certain other proportion of them have attained considerably more than 21 pounds at that age. The difference, then, between these extremes which go to make up the average, let us say, of 21 pounds at 1 year old, depends not only on their environment and their feeding, but also on whatever accidents of ill health have happened during the year.

It is important, I think, to differentiate between the breast-fed and the bottle-fed baby as to its weight, at certain periods, at least, of the first year. The normal breast-fed baby goes ahead very rapidly at first if feeding is successful, and is more likely to slow down during the latter part of the year; whereas, the bottle-fed baby, who has difficulty in accommodating itself to the artificial feeding, goes slowly at first, then shows a tendency, under the proper adaptation of its food, to gain more rapidly in the later months than perhaps the breast-fed child.

The breast-fed child shows certain tendencies to relax its gains. One of them, I think, was shown on one of the charts—Dr. Freeman spoke of it—

to be somewhere about the third or fourth month, when the maternal milk very frequently becomes relatively insufficient for the weight which the child has then attained, and when either attention to the mother or to her diet or giving supplementary feedings to the child is necessary in order to keep up a maximum gain. Another period which is shown on many of the weight charts is that when there is a flattening down of the curve during the last six months. This is a very common time for that relative insufficiency of the breast-milk, which must be supplemented, and can readily be supplemented, by artificial feeding, with the prevention of this flattening out and a more continuous gain. It is, therefore, of the highest importance that every child should be carefully supervised during its first year; because unless the child is definitely ill, it is often possible to keep it gaining more rapidly and adding more pounds or more ounces in weight than would be possible without supervision, without care of its diet and without the addition to its food whenever there seems to be a tendency to fall a little below the normal gain.

I also believe that the avoidance of the infectious cold is a very important thing for children during the first year—keeping them entirely away from these respiratory infections which so frequently cause a temporary loss of weight, or, at least, a very definite standstill.

DR. PISEK: It is a deplorable fact that we have not enough accurate figures of the weights and height of our American children. Particularly is this so between the ages of 2 and 5. I look forward to the time when this society will adopt a resolution that each member contribute his quota of measurements, all gauged along the one line, for example, following the plan outlined by the Congress of Anthropologists at Geneva—taking their standards and collecting a prescribed number of cases. Then we would not have to compare to the one unit (as Dr. Freeman has been obliged to do) with such large numbers as 96,000 compared with some hundreds. These indeed are valuable, and we need them; but we should have a collective investigation by this society; and I hope that it will come in the near future.

DR. SHAW: I agree with Dr. Pisek that we need reliable statistics. While we have not had any in the past in this country, I think in the state of New York we are going to be almost surfeited with them; because the legislature a year ago passed a law requiring every schoolchild to have a physical examination. The State Department of Education furnishes a blank which is to be filled by the school physician, or by the health officer; and the physician himself has to weigh and measure the child. As there are 2,000,000 schoolchildren under the department of education—that is, outside of New York—in a few years we will have a good many schoolchildren to get average statistics from.

I rather hoped that Dr. Pisek would say something about the scorecard which has been prepared by a subcommittee of the American Medical Association, which is called "The Standard Score-Card of the American Medical Association." Until my attention was called to the better babies contests last summer, I had no idea of the extent to which this is being carried on, more especially in the far West, and quite a large number in the eastern part of this country. It was found that there were between fifty and sixty different score-cards used in these contests. The Women's Public Health Committee of the American Medical Association felt that there should be a standard card, so that these various statistics could be correlated. I would suggest that if the members of this society do not wish to take the time to make the measurements suggested by Dr. Pisek, according to the outline prepared by the Smith-

sonian Institution, they could take such measurements as are outlined on this standard score-card, which are very simple, and, if they are taken by men of standing we would have some statistics that would be quite valuable and very easy to correlate.

DR. PISEK: I hope we will not accept the figures Dr. Shaw has given us. I say that advisedly, for this reason: we could not afford to accept figures that were indiscriminately made; such figures are bound to be erroneous. Figures that this society could accept must be made according to absolute standards, following a specific plan, using instruments of precision which are guaranteed by the Smithsonian Institution.

DR. FREEMAN: I would like to point out one fact that I believe to be true of the normal weight during the first year; that is, that the average gain during the first three months is about 5 pounds; during the second three months, 3½; during the third, 3, and during the last three months, about 2½ pounds. That is, the child gains rapidly during the first three months and somewhat more slowly during each succeeding three months.

DISEASES CONNECTED WITH MECKEL'S DIVERTICULUM

WITH ESPECIAL REFERENCE TO DIVERTICULITIS

J. P. CROZER GRIFFITH, M.D.
Professor of Pediatrics in the University of Pennsylvania
PHILADELPHIA

There occurred in my practice not long ago a case in which the combination of clinical manifestations and pathological lesions was so unusual, so unexpected, and so misleading that it seems worthy of being recorded. It belongs in the category of diverticulitis, but seems almost unique in some respects. At least I have failed to find any case on record giving just the same symptom-complex.

CASE REPORT

History.—Joseph C.; aged 19 months when first seen by me, March 15, 1912. The history given was that the boy had been in good health and well nourished until in the preceding summer when he suffered from a condition which his physician called "auto-intoxication"; although he later spoke of "scurvy." Possibly both disorders had been experienced. He recovered and remained well until November, 1911, when he had a fall downstairs, but apparently without discoverable serious injury. A week later he began to suffer from slight attacks of indigestion. Early in January, 1912, the mother was alarmed to find on one occasion that the formed stool which the baby had passed was of a partly reddish color, and that when put into water it stained it a deep red. Previous to this there had been some constipation. The bowels now remained normal and without constipation for two weeks, when another reddish stool was passed similar to the one described. The child suffered also from attacks of abdominal pain which were supposed to depend on indigestion. February 16 he had an attack of complete suppression of the urine lasting for over twenty-four hours. Catheterization showed the bladder to be empty. He then urinated very little for almost a week. There had been some slight drowsiness before the attack. There was no edema or nervous symptoms of any sort. At this time the mother noticed the change in the child's appearance, the former healthy tint having been replaced by a waxy whiteness which never afterward improved. After recovery from the suppression and oliguria, the attacks of paroxysmal abdominal pain continued to occur with greater frequency and constantly increasing severity, the child, when suffering most, often lying on the abdomen with the knees drawn up under him. Occasionally there was vomiting of thickly curdled milk. Constipation became troublesome and bowel movements were obtained only by the use of purgatives, the expulsion of feces being accompanied by much flatus. A Philadelphia pediatrist was called in consultation and made the diagnosis of anemia, which a blood-examination showed to be apparently of a

secondary nature. Nothing was said to the parents of the importance of the blood-colored stools. Probably the existence of such stools had not been mentioned by them. Repeatedly since the attack of suppression the feces had been of a liver-color or a reddish-brown. Nose-bleed had occurred once a few days before I saw him.

At the time of my first visit the baby was in a dull mental state, looked very ill, was strikingly pale, but was without edema or evident wasting. Abdominal pain was severe, and the abdomen moderately tympanitic and tender. Just before the visit there had been passed per rectum a small dark-red blood-clot the size of a pigeon's egg, and the child had also vomited. The great weakness of the patient prohibited satisfactory examination, and the only diagnosis possible seemed to be that of secondary anemia dependent on repeated, or, perhaps, almost continuous, intestinal hemorrhage, generally of small amount at any one time. The cause of the hemorrage was not certain, but ulceration of some portion of the intestinal tract seemed probable. The pain appeared to depend possibly on the tympanites, although this explanation was not entirely satisfactory. The significance of the attack of urinary suppression was not at all clear.

March 16.—There had been three or four intestinal hemorrhages of considerable size during the preceding night accompanied by much abdominal pain. The child was seen with me on this day by Dr. J. H. Jopson, attending surgeon to the Children's Hospital of Philadelphia, who could discover no rectal disease or anything abnormal in the abdominal cavity, although the tympanites prevented to some extent a satisfactory investigation. The question of exploratory laparotomy was discussed, but the uncertainty of the diagnosis and the extremely weak condition of the patient made this seem unadvisable. A 10 per cent. gelatin-solution was given freely by the mouth; albumin water prescribed as the diet, and laudanum in 2-drop doses ordered as demanded by the pain. A hypodermatic injection of 2 c.c. of horse-serum was administered by Dr. John Speese in the effort to control the hemorrhage.

March 17.—An examination of the blood made on this date by Dr. John Diven showed rapid coagulation on puncture; a hemoglobin percentage of 22; red blood-cells 1,590,000, and leukocytes 7,840. The differential count (300 cells) gave polymorphonuclears 76 per cent.; small lymphocytes 14 per cent.; large mononuclears 5 per cent.; transitionals 3 per cent.; eosinophils 1 per cent.; basophils 1 per cent. There was poikilocytosis, but no nucleated red cells were found. The examination of the urine made by Dr. C. A. Fife showed a specific gravity of 1.016; acid reaction; no albumin or urobilin; trace of indican; uric acid and phosphate crystals, no casts or red or white blood-cells.

March 20.—There had been no return of hemorrhage, and the pain was better without need for the laudanum. The general condition, likewise, had improved. The bowels had been opened daily by saline douches.

March 22.—The child was very much better, and was sitting up out of bed.

March 27.—Two days previously there had commenced a serious relapse in the condition of the patient, and the pain had been extremely severe; more than could in any way be accounted for by the tympanites. Some slight evidence of blood had appeared in the stools. Castor oil was administered on the day previous, and a very large mass of cheese-like consistency was passed, of a most offensive odor but apparently not containing blood. The child died exhausted on March 27. During the attack there had been a moderate irregular febrile reaction.

The final clinical diagnosis was that of secondary hemorrhage depending on ulceration somewhere in the intestinal tract. The cause of the severe pain was not clear. An autopsy was made by Dr. Howard C. Carpenter, whose report reads as follows:

Necropsy.—On opening the abdomen a localized peritonitis was seen just below the umbilicus. This localized peritonitis could have been placed in a circle having a diameter of 8 cm. In this area four coils of intestine were firmly matted together, and the serous coat of the bowel showed a fibrinous peritonitis. When the coils were forcibly separated an abscess was discovered which contained a little more than 2 drams of pus. Further dissection allowed the pus to escape and revealed a Meckel's diverticulum in the center of the abscess cavity. The diverticulum measured 3 cm. in length, and its walls were quite thick, averaging about 8 mm. but thinnest at the tip. It was firm on palpation, giving the sensation characteristic of a chronic inflammatory process. The mucosa of the diverticulum was swollen and eroded, and at the tip of the organ there was found an ulcer 8 mm. in diameter. The opening between the ileum and the diverticulum was small but patulous. The distal extremity of the diverticulum was free. There was a purulent exudate at its tip on the serous surface, corresponding in location to the ulcer on the mucous surface. The remainder of the intestine, including the appendix vermiformis, appeared normal. The mesenteric glands were somewhat enlarged, but without evidence of suppuration. The liver was normal in size but yellowish in color, showing a fatty infiltration. There were no metastatic abscesses discovered. The spleen was normal. The kidneys showed an acute diffuse nephritis. They were moderately enlarged and red in color, with a wide cortex containing reddish streaks.

DISCUSSION OF CASE

We have here, then, probably primarily an ulcerative inflammation of Meckel's diverticulum which was the cause of the persistent leaking of blood from the bowel, and of the severe anemia. As time elapsed, this inflammation extended to the serous layer of the diverticulum, producing a secondary, purulent peritonitis, localized by the matting of the coils of the ileum around the seat of suppuration. The severe abdominal pain was due to this peritonitis, and in part, perhaps, to the kinking of the intestine, making evacuation of the bowel difficult, and accounting for the constipation. The origin and bearing of the nephritis was uncertain. It appeared to have had little part in the final complex of symptoms and the fatal termination.

Meckel's diverticulum, the remains of the omphalo-mesenteric duct, although bearing the name of this investigator, was in reality, according to Fitz,[16] known earlier, being illustrated by Ruysch[41] in 1701. The organ may cause disease in various ways. The subject has been studied in its various aspects by numerous writers, among whom I may mention Rayer,[37] Fitz,[16] Kelynack,[27] Payr,[35] Hilgenreiner,[23, 24] Porter,[36] Bunts,[5] Turner,[46] Clogg,[10, 11] Forgue and Riche,[17]

Gray,[19, 20] Carnett,[7] Drummond,[14] Hertzler and Gibson,[22] and Wellington.[47] I shall, therefore, make no attempt at any exhaustive reference to the literature of the subject with details of reported cases; and those interested are referred to the list of writers, far from complete, found at the close of this contribution.

None of the affections of Meckel's diverticulum appear to be of common occurrence, although a persistence of the organ in some form is found in from 1 per cent. to 2 per cent. of all individuals. Thus Kelynack[27] found the diverticulum present in 13 out of 1,248 subjects examined (i. e. about 1 per cent.); the Committee of the Anatomical Society of Great Britain and Ireland[12] 16 times in 769 bodies (i. e. a little over 2 per cent.); and Mitchell[34] 39 times in 1,635 bodies. Hilgenreiner[23] gives statistics on 4,848 autopsies, reported by a number of investigators, with 90 having a diverticulum, i. e., about 1 in every 54 persons; and Bienvenue[3] in a similar manner analyzed a series of autopsy statistics and found 126 instances of the persistence of Meckel's diverticulum in 9,672 autopsies, i. e., 1.7 per cent. The organ is present much more frequently in males. Of Mitchell's[34] 39 cases, 34 were in the male sex. In Turner's[46] 145 cases in which the diverticulum was merely a post-mortem finding, without pathological alteration, the distal extremity was attached to some region in only 9 instances. On the other hand, in 161 cases with pathological changes it was attached in 110.

As a rule the presence of a Meckel's diverticulum gives rise to no symptoms whatever, and this is particularly true, as has just been pointed out, when it lies distally free in the abdominal cavity. A number of series of cases have been published, however, in which the organ occasioned symptoms of disease. Thus Porter[36] details 184 collected cases of disease of the diverticulum, although he found others on record with too meager a description to render them of value for study. Forgue and Riche[17] collected 287 cases of intestinal obstruction by a Meckel's diverticulum; and Wellington[47] 326 cases of disease of the organ. The largest series of reported cases of pathological conditions of the diverticulum appears to be that by Rostowzew[39, 40] of 634 cases. I have not had access to his original publications.

Lesions due to Meckel's diverticulum seem oftenest to produce symptoms in adult life, the average age of Porter's[36] cases being 21 years and 9 months. Bienvenue,[3] however, thinks the greatest number of pathological changes occur in childhood. Males are more fre-

quently affected than females; in 265 of Wellington's cases in which the sex was mentioned, there were 201 males and 64 females.

1. The most frequent lesion found is *strangulation of the intestine by the diverticulum or its remains,* usually attached to the umbilicus or to some part of the intestine, mesentery, or other region. In cases of obstruction by Meckel's diverticulum, Hilgenreiner[23] found the organ attached distally in the proportion of about 4 attached to 1 free, with the attachment most frequently to the mesentery. The diverticulum may either be in the form of a fibrous, cord-like remainder of the organ, or have its lumen still present, through all or part of its extent. In either case the ileum becomes ensnared in various ways, causing constriction and strangulation. Although a considerable number of such cases are reported in medical literature, the lesion is yet uncommon enough to produce probably a sensation of surprise in any physician discovering it. My own experience is limited to a single case made certain by autopsy. Of Porter's[36] 184 cases of obstruction by Meckel's diverticulum, 101 were due to the presence of a constricting band; and this was true, likewise, in 144 of Wellington's[47] 326 cases, not including intussusception of the diverticulum or volvulus.

The symptoms are those characteristic of most varieties of intestinal obstruction, with the exception of ordinary ileo-cecal intussusception, and it is only by operative interference or at autopsy that the diagnosis can be made with certainty. Intestinal obstruction due to this cause may occur in early life or later. There has been a division of opinion as to which period is more common. Forgue and Riche[17] and Hilgenreiner[23] agree that the most frequent age is from 15 to 25 years, and the weight of opinion seems to be in favor of this view. It is estimated by Halstead,[21] based on 991 collected cases of strangulation of the intestine from different causes, that 6 per cent. are dependent on Meckel's diverticulum. However, of the 669 cases of obstruction by bands occurring in the London Hospital during thirteen years, (Barnard[1]) but 21 (3.14 per cent.) were due to the diverticulum.

It is to be remembered that strangulation of the ileum by a Meckel's diverticulum is in a way retro-active, and the diverticulum in its turn, if it has retained its original character, may become strangulated by the ileum.

2. Another condition, properly a malformation, and naturally always congenital, is the persistence of Meckel's diverticulum with an *opening at the umbilicus.* This is unusual, and the subjects are nearly always males. Wellington[47] found but 21 instances in his 326 cases of disease of the diverticulum. Strasser's[43] statistics included a larger number, 63 in all. The lesion results in a fecal fistula through which feces may be discharged; or even, as in some reported cases, intestinal worms. The mucous membrane of the diverticulum protrudes at the umbilicus, forming a tumor, generally small, sometimes of larger size. The fistula is single and centrally situated; or sometimes, if the tumor is very large, there are two lateral openings. Occasionally the intestine may protrude and become strangulated. Sometimes a persistent Meckel's diverticulum is obliterated near the intestine, and in this event mucus, but no feces, is discharged from the fistulous umbilical opening.

3. Among other unusual conditions sometimes found is the *formation of a cystic tumor,* the diverticulum being obliterated at both ends, as in the cases reported by Fox,[18] and by Cawardine.[8] In the latter there was a cystic dilatation of the diverticulum in a new-born infant, filled with meconium.

4. *Concretions* of smaller or larger size may be present in the diverticulum. This was well illustrated in the case of Beach[2] in which there was a fecal concretion the size of a pigeon's egg, with communication through the diverticulum between the intestine and the bladder. In an autopsy reported by Sherren[42] many small black calculi were found in the diverticulum. Similarly, as in the case of the appendix, foreign bodies of various sorts have been reported present.

5. A very unusual condition is described by Bland Sutton[44] in which there is a *super-involution of the diverticulum,* as a result of which narrowing of the intestine is produced, the obliterative process in the diverticulum having passed beyond this region to the ileum.

6. In other cases a stenosis of the ileum may be caused by *traction* of a short diverticulum attached at its distal extremity or elsewhere.

A Meckel's diverticulum persisting as a short, free organ may give rise to trouble in various ways:

7. Here is to be mentioned the rare *invagination of the diverticulum,* which is very liable to be followed by an ileocecal intussusception. Cheyne[9] collected 16 reported cases of such intussusception. Twenty

of Porter's[36] 184 cases were intussusception, and 59 of Wellington's[47] 326. Hertzler and Gibson[22] have recently published a careful review of the subject with the details in abstract of 41 collected cases, omitting others not sufficiently well-reported to permit of study. The average age was 13 years; in sharp contrast to the early age at which simple intussusception of the bowel usually occurs. Inasmuch as the invagination of the diverticulum takes place on the lateral aspect of the ileum, it may not occasion complete obstruction, but merely project into the lumen of the bowel and cause hemorrhage. This hemorrhage is usually slight, and there are none of the symptoms of diverticulitis pointing to this disorder. In other cases intussusception of the bowel promptly follows that of the diverticulum.

8. Still less common than intussusception is *volvulus of the diverticulum, or of the ileum,* depending on the presence of this organ. But 8 of Porter's[36] 184 cases were of this nature. Wellington[47] gives in 326 cases, 9 of volvulus of the intestine dependent on Meckel's diverticulum. Very rarely there may be volvulus of the diverticulum without twisting of the bowel. This was seen, for instance, in a case reported by Fox,[18] and in one by Taylor.[45] Cawardine's[8] case of cystic dilatation already mentioned was produced by a volvulus of the base of the diverticulum occurring during fetal life. Volvulus of the diverticulum or of the ileum is more liable to take place if the distal extremity of the diverticulum is attached.

9. *Hernia* of the diverticulum is occasionally seen. This has been studied by Ekehorn[15] who collected 22 cases; and later by Gray[20] who has a list from literature of 42 cases. There were others found by Gray making the number 60 in all, but too briefly reported to be of value for study. Thirty of his cases were inguinal and 12 femoral. The average age was 37 years.

10. *Inflammation of Meckel's diverticulum*—the subject under especial consideration in this contribution—appears to have first been named "diverticulitis" by Blanc[4] in recognition of the analogy which it bears to appendicitis. The first case on record, according to Bienvenue,[3] appears to have been reported by Littre[31] in 1700. The subject has secured meagre consideration or none at all in such textbooks on medicine or pediatrics as I have consulted. A number of special contributions, however, have appeared in journal-literature or in theses treating solely or largely of this condition. I may refer especially to the theses of H. Blanc,[4] Bienvenue,[3] and Ketteler;[28] the

volume of Forgue and Riche;[17] and the journal-contributions of Clogg;[10, 11] Rebentisch;[38] Hilgenreiner;[24] Denecke;[13] Meyer;[33] Cahier;[6] and Hübschmann.[25] Some of the other writers already mentioned have discussed it to a limited extent.

The condition is one of the more infrequent of abdominal diseases depending on Meckel's diverticulum. Ketteler[28] (1900) found 5 cases of diverticulitis; Denecke[13] (1902) 9; Rostowzew[39, 40] (1902) 45; Porter[36] (1905) 17; Cahier[6] (1906) 39; Forgue and Riche[17] (1907) 59; Bienvenue[3] (1912) 40; and Wellington[47] (1913) 50. The variation in the reports depends in part on the date of publication; in part on the limitations placed by the authors. It is interesting to note the relative tendencies, respectively, of the vermiform appendix to exhibit inflammation, and of the diverticulum to produce obstruction. Thus Leichtenstern[30] could collect but 36 cases of occlusion of the intestine by the appendix, while he found 70 cases of occlusion by the more rare Meckel's diverticulum. Inflammation of the appendix, on the other hand, is one of the most common disorders, while that of the diverticulum is of very exceptional occurrence.

The distinction is to be made at the outset between primary and secondary diverticulitis. In the latter some other lesion develops first, as when the diverticulum constricts the ileum and is in turn constricted by it, with consequent inflammation; or as when the diverticulum is strangulated in a hernial sac. In the matter of primary diverticulitis a further distinction is to be made between acute and chronic or recurring cases, and the relationship of diverticulitis to obstruction of the bowel is to be studied. Bienvenue,[3] the author of one of the latest and, perhaps, the most extensive discussion of diverticulitis, gives as instances of chronic inflammation of the diverticulum those cases in which adhesion of the distal end of the diverticulum elsewhere than the umbilicus has at some previous time formed, as a result, he believes, of an earlier localized inflammation. On this basis he considers that the majority of instances of intestinal obstruction by the diverticulum acting as a constricting band are to be viewed as secondary to diverticulitis. Others, however, among them Forgue and Riche,[17] do not consider that these abnormal adhesions are necessarily or even usually the result of an earlier inflammation. However this may be, the chronic and the acute diverticulitis as now under consideration are quite different from this condition and are rarely the cause of a complete intestinal occlusion.

Etiology.—The causes of diverticulitis are obscure. Infection, of course, lies at the basis of the lesion. Previous digestive disturbances appear to have some bearing, and trauma, too, has seemed to have been causative in several instances, as in those of Hübschmann,[25] Kramer[29] and a few others. It should be remarked, however, that Hübschmann does not consider his case an instance of primary inflammation of Meckel's diverticulum, but of the formation of a peptic ulcer in it, following a fissure produced by the trauma, with subsequent perforative peritonitis. The influence of foreign bodies is uncertain. They have been found in a number of instances; but, as with appendicitis, they are not necessarily the cause of the inflammation. This is true also of the occasional discovery of intestinal worms in the diverticulum in cases of inflammation. Cahier[6] found foreign bodies present in 9 of 29 collected cases. Although the disease can occur at any age, the larger number appear to have developed after childhood is passed. Meyer[33] places the age of the majority of individuals at from 35 to 70 years; and of Cahier's[6] 36 cases 16 individuals were from 20 to 54 years of age, one-third of the number being children.

Pathology.—From the point of view of pathology diverticulitis is very analogous to appendicitis, and no detailed description of the lesions is necessary. As in appendicitis all grades of inflammation exist, extending from a mere catarrhal process, to gangrene and perforation with localized or general peritonitis. There may be perforation from ulceration without gangrene. This is oftenest the result of typhoid fever. Wellington[47] collected 6 instances of this occurrence, and 2 from trauma. Makins[32] found a diverticulum with 13 perforations. Ulceration from any source is, however, uncommon.

Symptoms.—The symptoms vary to a certain extent within wide limits. Cahier[6] found 10 chronic cases with acute recurrence out of 37 instances of diverticulitis. In these more subacute or chronic or recurring cases there exist before the final crisis evidences of gastric disturbances, constipation, and recurrent painful attacks oftenest in the region to the right of the umbilicus or in the right iliac fossa. In the primarily acute cases, or in those with an acute development consecutive to the more chronic condition, the symptoms so closely simulate those of acute appendicitis that the diagnosis of this latter condition is generally made. The earliest stages of the disease may be entirely without clinical manifestations. In well-developed cases the symptoms consist in severe abdominal pain, which is liable to be

higher and nearer the umbilicus than in appendicitis; abdominal tenderness and resistance, and, finally, dulness on percussion in this region; nausea and vomiting; fever; and leukocytosis. Constipation is a frequent symptom, but diarrhea is as often seen. In a few cases of a subacute or chronic nature, such as those of Hilgenreiner,[24] Hübschmann,[25] Meyer,[33] and my own, passage of blood from the bowel took place. My own case was most unusual in that the intestinal hemorrhage was for a time the most marked symptom, and was certainly one of the decided causes of the fatal ending. Finally, in diverticulitis there may develop the symptoms of a general peritonitis, as in cases of appendicitis. This is more liable to occur than in the latter affection.

The question of intestinal obstruction as a symptom of diverticulitis has already been alluded to. It is possible that obstruction may become complete from the compression of the intestine, if the diverticulum is distally adherent, or from inflammatory adhesions or kinking, or in other ways. This is, however, exceptional. More or less difficulty in the evacuation of the bowels is liable to occur in any case of diverticulitis, either from the mere kinking of the coils of the intestine involved in a localized peritonitis, or as a result of paralysis of peristalsis, as seen sometimes in appendicitis. How important a factor this latter may be is uncertain. Cahier,[6] for instance, believes thoroughly in its existence, while Meyer[33] thinks it plays little part. In cases of secondary diverticulitis, complete obstruction may first take place, and later evidences of diverticulitis may develop, finally with localized or general peritonitis.

Diagnosis.—The question of the diagnosis of diverticulitis is of great interest. Is such a diagnosis possible? It is to be noted that the lesion has never been correctly diagnosed during life, although in a case reported by Karajan[26] it would seem that this might have been made. In this instance there were the symptoms suggesting appendicitis, but occurring in a man who had had in infancy up to the age of 3 years a fistulous opening of Meckel's diverticulum at the umbilicus. In general the study of diagnosis leads mostly to discouragement. Certainly, at least the effort to come to a correct conclusion should be made. The fact that the localized inflammation of a diverticulitis is situated oftenest in the lower part of the abdomen and toward the right makes the diagnosis of appendicitis more probable; and, as a matter of fact, most cases of diverticulitis have been thus diagnosed,

and the true condition discovered only at operation. In a case reported by Hilgenreiner[23] both diverticulitis and appendicitis were present. The origin of the diverticulum, oftenest at from 1 to 3 feet above the cecum, and its attachment to a freely movable portion of the intestine, make its position in the abdominal cavity a very variable one, and the localization of symptoms is consequently subject to no fixed rule. In the cases of secondary diverticulitis in which complete intestinal obstruction has already occurred, the development of inflammatory symptoms is suggestive but not conclusive, since any form of intestinal obstruction may be followed by signs of peritoneal inflammation. The presence of blood in the stools, although of rare occurrence, is, when present, suggestive of diverticulitis; yet this, too, is not conclusive, inasmuch as the symptom is in no way pathognomonic. The diagnostic features as based on the cases reported by different writers may be summarized as follows:

1. Localization of the pain and tenderness not so often at MacBurney's point as somewhat higher and to the right of the umbilicus, or even about it, or in some entirely different region.

2. An area of puffiness or of firm resistance in this region.

3. Absence or slight degree of meteorism, at least early in the attack.

4. The presence of blood in the stools and in the vomited matter.

5. The earlier existence of an umbilical fistula, or of some malformation elsewhere in the body.

Prognosis.—As regards the prognosis, it is impossible to reach accurate conclusions, due to the fact that catarrhal diverticulitis, recovering, has never been recognized during life; and it is impossible to determine how often such a condition has existed. That a diverticulitis of this sort with recovery does actually occur is shown by operations and autopsies, where evidence of former inflammation has been found. The prognosis of acute severer cases is unfavorable. Cahier[6] found a mortality of 65 per cent. in 35 cases, all those recovering having been operated on. In 50 cases of Forgue and Riche's[17] list the mortality was 53 per cent. Wellington[47] estimated the mortality at 40 per cent.

Little need be said of treatment. The only legitimate one is operative. Insasmuch as even a healthy diverticulum is a much greater and more constant menace to life than a vermiform appendix in a similar condition, a Meckel's diverticulum found in the course of an abdominal operation should be removed.

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THE ARTIFICIAL COOLING OF SICK-ROOMS IN SUMMER

A PRELIMINARY REPORT *

HENRY HEIMAN, M.D.
NEW YORK

The mortality of gastro-enteritis in infants, despite all scientific efforts devoted to its amelioration, is so appalling, that each summer, in addition to employing what is best in the old methods of treatment, we attempt to devise newer and better ways of combating this formidable disease. It was with this in view that during my summer service of 1913, an endeavor was made to attack this malady in a way entirely different from any previously undertaken.

The etiology of gastro-enteritis has been and will be for years to come a much discussed subject. Without reiterating the well known conclusions of many pediatrists, based on theoretical considerations and practical observations, let us state that in our opinion also, gastro-enteritis is probably primarily a nutritional disorder, having as a secondary causal factor a bacteriologic basis. Both of these factors operate in winter as well as in summer. To account, then, for the marked prevalence of diarrheal diseases in summer we must consider a third potent cause, namely, the heat.

In order to determine the exact part heat really plays in the production of these summer diarrheas, and the effect of treatment in a routine manner with this important factor eliminated, a cooling plant was installed at the suggestion of Dr. Goldwater, superintendent of the hospital, in one small ward of the children's pavilion whereby the temperature of the room was kept constantly lower than that of the outside atmosphere. In brief, the method of cooling was as follows:

Outdoor air is forced by a motor-driven Sirocco fan through a water-chamber in which the water is cooled by brine coils. From this chamber the air is forced through an upper chamber filled with additional brine coils, thence through short ducts into the wards, where the cooled air enters near the floor level. Open transoms allow the warmer strata of vitiated air to

* From the Children's Service of Dr. Henry Koplik, Mt. Sinai Hospital, New York.

escape from the top of the rooms. Some air also escapes through the entrance door when used by the nurse and the physician. The cooling plant proper is located in the basement under room No. 1, as shown on plan, Figure 1. It consists of a Greef air washer, a motor-driven blower and coils through which cold brine is forced, an eliminator for removing the free particles of moisture and heating coils for reheating the air, if so desired.[1]

The temperature was recorded by means of two recording thermometers, one registering the outdoor temperature, the other that of the room. The relative humidity was ascertained by a sling psychrometer. Observations of the temperature and relative humidity were also taken in the general ward where no cooling was done.

Typical charts of the recording thermometers are reproduced in Figure 2, showing the records for the hours from 9 a. m., July 31,

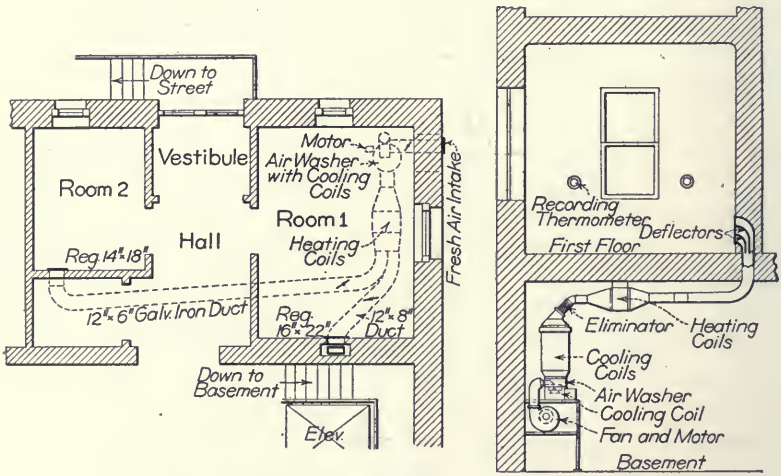


Fig. 1.—Location and details of air-cooling plant.

1913. The outdoor temperature, as will be noticed, was 86 F. at 9 a.m., rising gradually to an average of 92 between 1:30 and 4: p. m., then dropping gradually to 78 between 5 p. m. and 6:30 p. m., when it began to rise again. The indoor temperature of the room was maintained at an average of 72 between 9 a. m. and 3 p. m., rising gradually to 74 at 7 p. m., which latter temperature was maintained until midnight, then rising to 75 until 4 p. m., dropping again to 74 until 7 a. m. and 72 at 8:45 a. m. The temperature in the cooled

1. From a description by A. M. Feldman, M. E., Consulting Engineer, in the Proceedings of the American Society of Heating and Ventilating Engineers.

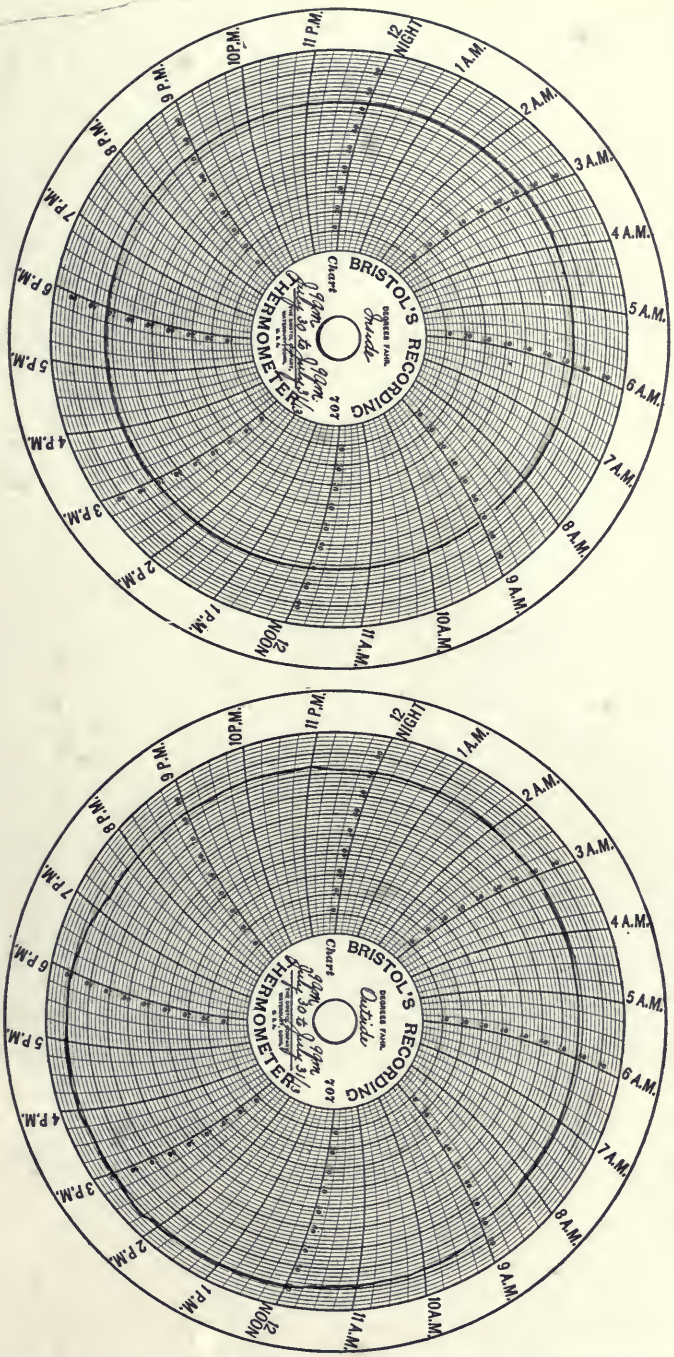


Fig. 2.—Charts showing record of inside and outside temperatures, July 30 and 31.

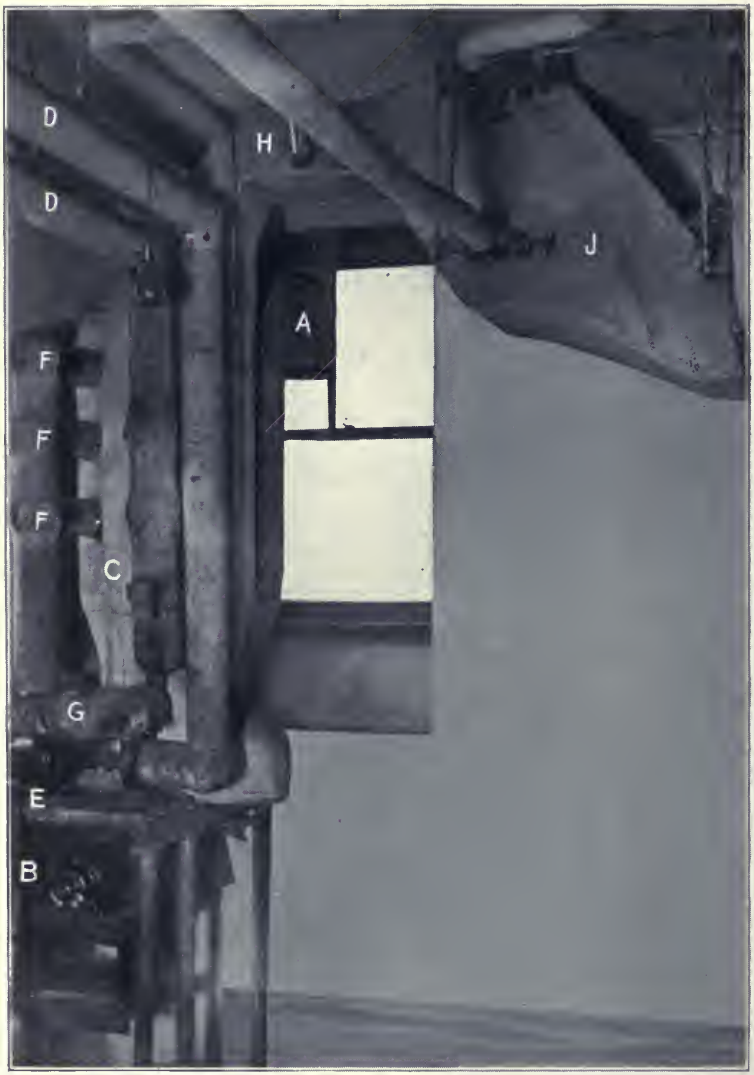


Fig. 3.—View of the air washing and cooling apparatus. The fresh air enters the duct at the point A in the upper sash of the window. At B may be seen the blower which delivers to the cooler-washer and cooling coils in chamber C. The pipes D D, are brine pipes tapping the return brine line of the general refrigerating system of the building. At E is the small pump for securing a positive circulation of the brine. The brine pump delivers to the washer through the pipe G and to the three coils above the washer F, F, F. At H is the delivery duct from the air washer and J is the reheating chamber, fitted, as indicated, with thermostatic control, the reheating coil also divided into three sections and the sections having hand valve control in the usual way. Incidentally the brine pipes are provided with thermometer wells and a thermometer is shown at the side of the duct to obtain readings of the air on delivery from the air-washer cooling system. A thermometer was also arranged for on the delivery side of the reheating chamber, so that arrangements are available for making a comprehensive test.

room varied during the period of observation from 63 to 74. This latter temperature was reached only on one day when the temperature on the street rose to 93. The average temperature in the cold room for 252 readings was 69 during a period of forty-two days. The records of the outdoor temperature during the same forty-two days, taken at 8 a. m., 12 m., 4 p. m., 8 p. m., 12 p. m. and 4 a. m., were 77, 79, 79, 80, 74 and 70 F., respectively. The temperature of the cold room was at all times from 5 to 11 degrees below that of the outside air.

The relative humidity was observed at different intervals during the day. In the artificially cooled room, the relative humidity varied between 41 and 76, making an average of 60 for readings taken on twenty-five different days. In the main ward, the relative humidity varied during the same period from 35 to 80, with an average of 59.

It is interesting to note that while low temperatures were obtained in the cold room, the relative humidity was approximately the same percentage as that in the main ward with higher temperature, thus indicating that the absolute humidity was reduced by the process of cooling.

Thirteen selected cases were treated, the average period of treatment being about two weeks. By selected cases is meant patients suffering from gastro-enteritis, ruling out premature and moribund infants. Six of the thirteen were in poor condition at the time of admission. One of these died shortly after entering the hospital. All the others were discharged from the hospital well. The treatment in all these cases was not different from that ordinarily carried out; this included *Eiweissmilch,* skimmed milk or barley water, plus the usual symptomatic medication.

Inasmuch as only thirteen cases of gastro-intestinal diseases were treated in the artificially cooled ward, it is hardly justifiable to present definite conclusions as to the therapeutic value of this measure. Gastro-enteritis was not particularly prevalent during the summer of 1913, and the number of cases treated in the hospital was very much below the number usually treated; in fact, the thirteen patients who were treated in the cooled ward were practically all the gastro-enteritis cases available for our first tentative studies. Thus there was no opportunity for comparison of the behavior of these cases with that of patients treated for the same disease in the open wards, under ordinary conditions. While unable at the present time to draw

far-reaching conclusions, we have nevertheless gained the impression that the babies treated in this ward were, on the whole, more comfortable, cried less often, looked less sick and less parched, rested and slept better than the patients kept in the other wards of the children's service during the same time.

It is not our intention to belittle the present methods of treatment of this disease, as well as the efforts of the Board of Health, the Social Welfare Department, milk depots and other associations for the prevention of infant mortality, but simply to introduce this additional therapeutic measure as a valuable adjuvant in the eradication of this malady. The only cases in which this method may be harmful are in weaklings, premature and moribund infants, in whose cases extreme heat is a powerful factor in sustaining life. In all other cases of gastro-enteritis, no matter how severe, the artificial cooling of the sick-room will in our opinion prove to be an extremely helpful measure.

I hope to have the opportunity of studying this important question further this summer. I wish to thank Dr. Louis H. Levy and Dr. A. Brody for their kind assistance in carrying out these observations.

30 West Eighty-Eighth Street.

DISCUSSION

DR. BOWDITCH: I should like to give you a little assurance from the experience that we have had in the Boston Floating Hospital, and point out possible pitfalls. I think one of the best things that I noticed in the apparatus shown was a short air duct. We unfortunately had long ducts. Our fans were at the end of a long chain of ducts. The amount of dust taken out of those ducts at the end of the year was tremendous. It clogged the machine, and made it practically impossible to get proper ventilation. I should think that a series of such apparatus placed near the different wards, would be far better than having a central supply-plant. We rely now on through and through ventilation. The windows are brought down to the level of the wainscoting.

DR. VEEDER: We have built one room in the St. Louis Children's Hospital practically like a refrigerator. It is piped on the ceiling and one side and is built in with non-conducting walls and ventilation provided, the idea being to reduce the entire temperature of the room without having cold air coming in.

DR. ADAMS: Some fifteen or more years ago when a baby was sick with summer diarrhea in a private residence in which the thermometer was day and night above 90 F., I conceived the idea of directing a current of air from the electric fan across a 200-pound cake of ice, with the effect that the temperature of that room was kept down to 75 or 80 F. The humidity was not tested; but this fact was illustrated: The child who had been in a profound coma, came to from the cooling—but we thought possibly a catarrhal condition had developed and the mother became frightened and we had to stop the fan, and the

child lapsed into coma as soon as the temperature jumped to 95 F. I took the child to New York, and on the boat and going from Peekskill over to the Catskill mountains, I thought he would die in the car. It was 112 F. in the car and people began to fan, so as to keep the air in circulation, if possible. The child was absolutely in coma when put on the elevator. When he got up to the mountain top he asked me if he could go riding in the carriage, showing that the temperature had a very beneficial effect on him.

DR. SHAW: Some of the members of this society may have seen our five-dollar cooling plant in one of the outdoor wards at St. Margaret's, Albany. This is simply a long stretch of unbleached muslin, six feet wide, attached above to a perforated water pipe, while below a tin trough catches the water. The sheet, being kept constantly wet, cools the atmosphere several degrees and is very comfortable on hot days. This works very satisfactorily and is perfectly simple and extremely cheap.

DR. HEIMAN: In answer to Dr. Bowditch, I think he is absolutely right in reference to the disadvantage of the long ducts.

In regard to Dr. Talbot's question, I think he is also right, because our nurses suffered a good deal from cold feet; so that we intend to have them work on a raised platform, knowing that cold air sinks to the floor. The air that we give our children is absolutely pure: it is washed and filtered at the same time.

GAUCHER'S DISEASE, WITH A REPORT OF TWO CASES IN INFANCY

J. H. MASON KNOX, JR., M.D.
BALTIMORE
AND
H. ROSWELL WAHL, M.D.
CLEVELAND

The case we should like to report as being of unusual interest is that of S. G., white, female, 9 months of age, seen at The Thomas Wilson Sanitarium, during the summer of 1912.

Family History.—The father is aged 41 years, moderately alcoholic. The mother is 38 years of age and well. Both parents are Russian Hebrews of small means. There have been eleven children, aged from 20 to 4 years; six are apparently well. Three died in infancy, one from an unknown cause, one smothered and one of convulsions. A boy of 14 died of hip disease. The father's brother died of tuberculosis but did not live with the patient.

Past History.—The patient's birth was spontaneous. She was breast fed for one month and then given cow's milk mixture, under the direction of the Baby Welfare Station. She never thrived and did not grow like other children. For a month or two she vomited the milk a good deal, but for three months the milk was well retained. She slept well, cut two teeth at 6 months, and was never acutely sick. The gain in weight, however, has been very slow. The stools are two to four in twenty-four hours, yellow and well formed.

Physical Examination.—On admission to sanitarium the patient weighed 11 pounds. She was considerably emaciated. The skin was pale, with a slight brownish pigmentation. The abdomen was enlarged and the superficial veins distended. There was no general glandular enlargement, though the cervical and axillary glands were slightly hypertrophied. The lungs were clear. The heart was not enlarged. There was a soft systolic murmur heard at apex. The size of the abdomen was due to a great hypertrophy of liver and spleen. The upper border of liver began at the sixth rib and extended 2½ inches below the costal margin in the right nipple line. The left lobe was

palpable 2 inches below the sternum and extended 2 inches to the left of the middle line. The surface of the liver was smooth and firm, the notch readily palpable. The spleen was represented by a large mass extending 4½ inches below the left costal margin, to the level of the umbilicus. The surface was firm and a little roughened, the notch was readily palpable. The kidneys were not felt. The reflexes at the knee were present. The blood examination on admission was as follows:

Leukocytes	10,400
Red cells	5,200,000
Hemoglobin	90/100

Differential Count:

Polynuclear	42	per cent.
Large mononuclear	5	per cent.
Small mononuclear	41	per cent.
Transitionals	3½	per cent.

Examination of stools showed an excess of fatty acid crystals and no bile. There was little change in the child's condition in the first month after admission, excepting that she lost gradually in weight. At this time the blood condition was as follows:

Leukocytes	25,400	
Red cells	4,400,000	
Hemoglobin	60	per cent.

Differential Count:

Polynuclear	59	per cent.
Large mononuclear	3.2	per cent.
Small mononuclear	36	per cent.
Transitionals	1	per cent.
Myelocytes	1.02	per cent.

The patient continued to lose strength and weight during the last month of her life and died at the age of 11 months, two months after admission to the sanitarium. The day before death a marked change took place in the differential count of the leukocytes. Their total number was increased to 35,000. These were made up of

Small lymphocytes	82	per cent.
Large lymphocytes	9	per cent.
Large mononuclear	5	per cent.
Polynuclears	3	per cent.
Unclassified	1	per cent.

The clinical diagnosis, somewhat influenced by the last blood picture, was lymphatic leukemia, although previously Banti's disease was considered with hypertrophic cirrhosis of liver and spleen, and the possibility of the enlarged liver and spleen being due to secondary metastatic growth from underlying neoplasm was discussed.

Autopsy.—Autopsy was performed four hours after death. The principal gross changes were found in the liver and spleen. The surface of the liver was smooth and the capsule not thickened. Lobules appeared as minute pink centers with yellow peripheries. The organ cut with increased resistance, leaving a pale yellow moist surface from which a yellowish mucoid material, resembling condensed milk, could be scraped off. There was no congestion. Weight 415 gm.

The spleen had a mottled pale red color, smooth surface and a capsule that could easily be stripped off. The pulp on section was very friable and the same kind of material noticed in the liver was obtained in scraping the cut surface. Weight 86.5 gm.

The adrenal glands were enlarged and swollen.

The intestinal mucosa showed areas of congestion and enlarged Peyer's patches and solitary follicles. There was no ulceration.

There was a general enlargement of lymph glands throughout the body. They had a uniform appearance, were bright yellow in color and were rather firm in consistency. From most of the glands a viscid yellow material, like that found in the liver and spleen, escaped after incision.

Microscopic Examination.—Microscopic examination of the important tissues may be summarized:

Liver: The normal architecture of the liver lobule was much distorted; many of the capillaries were filled with blood cells. No endothelial lining could be distinguished. Two unusual types of cells were present; both were rounded in shape and appeared swollen, the cytoplasm was pale and filled with numerous small vacuoles; the nucleus was small and poorly differentiated.

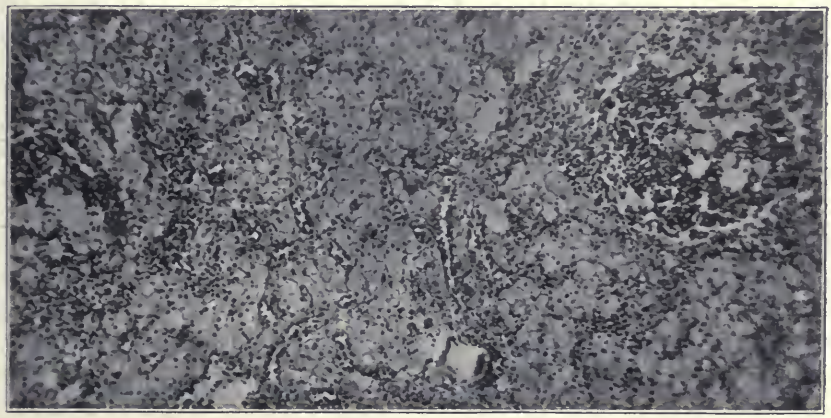
The other type was a little smaller and contained more cytoplasm and stained more deeply with eosin. These cells are more regularly and diffusely distributed throughout the lobule, the darker staining type predominating. No stains of fat globules were obtained.

Spleen: The most striking change was the relative absence of lymphoid and splenic pulp cells. All through the organ there were large rounded spaces occupied by more or less rounded swollen cells, with small nuclei and pale granular or vacuolated cytoplasm. There were a few irregular foci of lymphocytes and splenic cells among these very large cells. The lymph and blood spaces were dilated and the endothelial lining cells were considerably swollen.

Kidneys: The capsule was not thickened. The endothelial cells of Bowman's capsule were some of them swollen and showed a vacuolated cytoplasm resembling the large cells described in the spleen. There was no interstitial infiltration.

Adrenals: The capsule was a little thickened in the cortex, the cells appeared swollen with a tendency to undergo a vacuolar or edematous degeneration. The medullary portion was composed of large irregularly rounded, vacuolated cavities embedded in a coarse reticulum. These spaces were found to be enormously swollen edematous cells with a vacuolated cytoplasm, resembling the large cells found in the spleen.

Thymus Gland: There was a marked absence of lymphoid cells. Their place was taken by a wide meshed reticular tissue composed of large granular cells similar to those described in the spleen and adrenal gland.



Section of spleen magnified sixty-five times, showing the relative disappearance of the lymphoid cells in the pulp and the presence of many large, pale, granular or vacuolated cells between the sinusoids. Note similar large, pale cells within the malpighian bodies.

Lymph Glands: The lymphatic glands presented a variable appearance. In some there was a predominance of the lymphoid cells with a few scattered vacuolated cells similar to those described in the thymus and spleen; in others no lymphoid cells were seen, all being replaced by the large, rounded, vacuolated cells giving the gland the appearance more of adipose than of lymphoid tissue. All intermediate stages between these two types were found. The blood and lymph sinuses were markedly distended in some of the glands, the endothelial lining being swollen. Some of the large vacuolated cells seemed to be traceable to the endothelial cells; others seemed to be derived from the endothelial plate-like cells of the reticulum.

Gastro-Intestinal Tract: The cells of the superficial part of the mucosa showed an edematous degeneration resembling the cells described in the lymphoid tissues; the muscular coat was much hypertrophied. The cytoplasm of the muscle fibers often contains small vacuoles. Throughout the muscular coat there were foci of edematous clear areas composed of large vacuolated and

granular cells similar to those described in the spleen and lymph structures. Similar vacuolated cells were found in Peyer's patches.

Heart: Here, too, the muscle fibers appeared shrunken and atrophied and were the seat of a vacuolar and granular degeneration. In some areas, on cross section, the tissue presented the appearance of a markedly fatty infiltrated liver, scarcely to be recognized as heart tissue.

Lungs: The main change was a peculiar vacuolar swelling or edematous change in some of the cells of the alveoli walls.

Anatomical Diagnosis.—Gaucher type of primary splenomegaly—vacuolar degeneration of the parenchymatous tissues.

DISCUSSION

The essential lesion in the case is the invasion of various parts of the body, particularly the lymph-adenoid tissues by large polygonal or round faintly staining cells enclosed in a network. The cells seem to have undergone an edematous degeneration the exact nature of which is still undetermined. Moreover, the origin of these characteristic cells is also under discussion. The consensus of opinion seems to be that they arise either from endothelial cells or from normal reticulum in the blood-forming apparatus. Mandlebaum, who has made the most recent and satisfactory study of this condition, feels that these large cells may be divided from lymphocytes or reticulum cells in the germinal centers of the follicles. It is possible that the cells may have several origins. The disease described belongs to that type of primary splenomegaly first described by Gaucher[1] in 1882. Gaucher considered the large cells in the spleen and lymphoid tissues as epitheliomatous; that is, real tumor cells; but this view has been largely disproved. The literature has been carefully collected by Brill and Mandelbaum[2] up to December, 1913. They found thirteen cases proved by microscopic examination of the organs. The youngest was aged 2 years, the oldest 33 years. The disease appeared before the age of 12 years in the majority of cases.

Since this publication, three additional cases have been reported by Nieman,[3] Erdman and Moorhead[4] and by Herman, Roth and Bernstein.[5] These, with our cases, make a total of eighteen instances in which the condition was proven by microscopic examination. The case

1. Gaucher: These de Paris. 1882.
2. Brill and Mandlebaum: Am. Jour. Med. Sc., 1913 (Dec.).
3. Nieman: Jahrb. f. Kinderh., 1914, lxxvix, 1.
4. Erdman and Moorhead: Am. Jour. Med. Sc., 1914, cxlvii, 213.
5. Herrman, Roth and Bernstein: Arch. Pediat., 1914, xxxi, 340.

of Nieman, a girl 17 months of age, is apparently identical with those here reported. The disease often affects more than one child in a family, though there is no tendency to hereditary transmission. Seven additional cases have been described in the families of the proved cases, on the basis of clinical examination. The characteristic symptoms of the disease as outlined by Brill and Mandlebaum are its frequent presence in members of the family of the same generation, the progressive increase in the size of the spleen and later of the liver, a brownish, yellow discoloration of the skin, a yellowish wedge-shaped thickening of the conjunctivae commonly seen on both sides of the cornea, and the prolonged and chronic course of the disease, which may not for a long time disturb the health of the individual. In the early stages there is a probable leukopenia, but no very definite blood picture has been described. The anemia has been rarely pronounced in any stage. There is no jaundice and the course of the disease is not that of a malignant growth. The pathological feature is the presence in the spleen, liver, lymph glands and bone marrow of distinctive large cells with characteristic cytoplasm and small nuclei.

The case here reported presents some peculiarities possibly because of the extreme youth. The large vacuolated cells involve the *thymus, Peyer's patches* besides the spleen, lymph glands, bone marrow and liver as described in other cases. Another striking feature is the vacuolated degeneration present in all the parenchymatous cells of the body. In our case the large cells seem to be derived both from the endothelial cells of the capillaries and of the lymph-adenoid reticulum. They contain material that is not fat but seems to be a kind of lipoid matter, the nature of which is in doubt, but is probably an unusual degenerative product. The intestinal tract showed acute enteritis. It is possible that there may have been an acute vacuolar degeneration of the parenchymatous tissues due to intestinal intoxication superinduced on a Gaucher's type of primary splenomegaly. The process was probably older in the spleen and liver than elsewhere. Many of the lymph glands showed very early involvement, yet some of the glands are entirely devoid of lymphoid cells. So it is possible that it began in certain lymph glands and spleen simultaneously and then extended to other organs. The presence of similar cells in foci in the walls of the stomach and in the medulla of the adrenal gland suggests that while the process may be initiated in the hemopoietic system, it can extend to other organs, especially to those that have a fine reticular supporting meshwork.

No treatment has proved helpful. In a number of instances splenectomy has relieved the symptoms of abdominal pressure, but there is no evidence that it has caused an arrest of the process. The involvement of all the organs, as in our cases, shows clearly that no cure can be effected in extensively involved cases by removal of the spleen.

A confirmation of the tendency of the disease to recur in families came under observation while we were studying the sections of the case already described. The mother of the patient came to the clinic with another baby, 5 months of age, a girl with the same name and presenting a condition apparently identical with that of the sister who had died twenty months previously. The second child had never thrived, had always been malnutritic and pale and had some eczema.

The skin was a grayish yellow color, the right lung was clear, the left showed some dulness posteriorly, with a few moist râles. The abdomen was large, with distended superficial veins. The liver was markedly enlarged and firm, extending from the sixth rib to 2½ inches below the costal margin. The spleen was also greatly hypertrophied, the lower border reaching below the level of the umbilicus. Electrical reactions were normal. The blood was practically normal as follows:

Hemoglobin	65 per cent.
Red blood cells	3,024,000
White blood cells	11,200

Differential Count:

Polymorphonuclear cells	33 per cent.
Small mononuclear cells	41 per cent.
Large mononuclear cells	20 per cent.
Transitionals	2 per cent.
Eosinophils	3½ per cent.

There was noted in the blood examination a large excess of platelets, and an unusual number of fragmented cells and a few myeloblasts. Repeated blood examination showed but slight changes. The number of leukocytes at times exceeded 20,000. The patient's condition remained about the same. She took food badly and lay listlessly through the day. She had little rise in temperature. The von Pirquet test and the Wassermann reaction were negative. It would seem probable, therefore, that the second baby was also suffering from the same unusual condition. The cases together represent the only ones thus far described under 1 year of age.

The condition has been described under a variety of names. The terms including splenomegaly and hepatomegaly fail to indicate the involvement of other tissues. The condition is not primarily a disease of the spleen or of any other organ or set of organs, but is a general one due to a disturbance of fat metabolism, a kind of lipoid metamorphosis with formation of characteristic large pole cells. We believe that the name Gaucher's disease should be retained for the general condition until the nature of the disease is better understood.

[Note.—The patient has since come to autopsy and showed precisely identical lesions with the first case. Both cases will be more fully reported. Before death the diagnosis in the second case was confirmed by the examination of a superficial lymph gland in which the characteristic cells were found. Radium therapy applied to the spleen in the second case was followed by a marked reduction in the leukocytes in the circulation, 22,000 to 2,000 in a few days, but otherwise it produced no noticeable change.]

A CASE OF GAUCHER'S DISEASE IN A BOY THIRTEEN YEARS OF AGE. SPLENECTOMY WITH RECOVERY

CHARLES HERRMAN, M.D.
Attending Pediatrist to Lebanon Hospital

SURGICAL REPORT BY HENRY ROTH, M.D.
Attending Surgeon to Lebanon Hospital

PATHOLOGICAL REPORT BY EUGENE P. BERNSTEIN, M.D.
Pathologist to Lebanon Hospital
NEW YORK

The case here reported is the second of Gaucher's disease which has come under my personal observation. The first (Case 5 of Dr. Roth's table) was a boy, three and a half years of age, who came to my service at the Vanderbilt Clinic in 1911. The clinical history was reported by Dr. Reuben.[1] A splenectomy was performed by Dr. Wiener. The patient died shortly after the operation and a complete pathological report was published by Dr. Mandelbaum.[2]

The subject of Gaucher's disease has been so thoroughly discussed recently in an excellent monograph by Drs. Brill and Mandelbaum[3] that I shall confine myself to a brief clinical history of the present case.

The patient is a boy now fourteen years of age. His mother is healthy; his father had a gastric ulcer for which a gastro-enterostomy was performed. There are eight other children, all apparently healthy, except a sister of seven years, who has a tuberculous coxitis and an enlarged spleen, palpable 5 cm. below the free border of the ribs in the axillary line. It is noteworthy that the two children affected are the last of a large family. A similar peculiarity is seen in a certain percentage of cases of Mongolian imbecility. The patient had measles and whooping-cough, but no manifestations of syphilis, tuberculosis or malaria at any time. He first came under my observation in November, 1906, at the age of seven. The mother had noticed an enlargement of the abdomen when he was four years of age. At six and a half years he had severe abdominal pains and epistaxis. A physical examination

1. Reuben: Am. Jour. Dis. Child., 1912, iii, 28.
2. Mandelbaum: Jour. Exper. Med., 1912, xvi, 797.
3. Brill and Mandelbaum: Am. Jour. Med. Sc., 1913, cxlvi, 863.

showed a spleen extending 8 cm. below the level of the umbilicus and 1 cm. beyond the umbilicus in the median line. The edge of the liver was felt 5 cm. below the free border of the ribs in the right mammary line. The lymph nodes were not distinctly enlarged. Examination of the blood showed: Hemoglobin, 75 per cent.; red blood cells, 4,390,000; white blood cells, 4,000; polymorphonuclears, 70 per cent.; lymphocytes, 20 per cent.; eosinophils, 1 per cent. No plasmodia. Quinin, arsenic, iron, mercury and iodids were given at various times without any apparent effect. In June, 1909, he had a hemorrhage following the extraction of a tooth and in July an attack of severe abdominal pain followed by the vomiting of a large quantity of blood. On July 13, 1909, he was admitted to my service at the Lebanon Hospital and remained in the hospital one month. Physical examination revealed some discoloration of the face, a venous hum in the vessels of the neck, a functional murmur at the base of the heart, an enlarged abdomen measuring 58 cm. at the level of the umbilicus, which increased to 62 cm. during his one month's stay at the hospital. The liver extended from the fifth rib above to 5 cm. below the free border of the ribs in the right mammary line, 3 cm. in the right axillary line and 6 cm. in the parasternal line. The spleen extended from the seventh rib above to 15 cm. below the free border of the ribs in the left axillary line and 1.5 cm. beyond the umbilicus in the median line.

A series of examinations of the blood showed:

	Hemoglobin	Red Blood Cells	White Blood Cells	Poly-nuclears	cytes Lympho-
July 14, 1909	60 %	3,640,000	3,800	56 %	44 %
July 19, 1909	50 %	3,650,000	3,600	48 %	52 %
Aug. 3, 1909	55 %	3,620,000	3,400	52 %	48 %
Aug. 9, 1909	64 %	3,800,000	2,800	50 %	50 %

A few microcytes and slight poikilocytosis. The urine was acid, of a specific gravity varying from 1.010 to 1.020; no albumin or sugar. The examination of the feces showed nothing abnormal. He had occasional rises of temperature to 101°. He had three attacks of epistaxis, none very severe. His weight on admission was 52½ pounds and during his stay he gained 3½ pounds. He received seven x-ray treatments at intervals of three or four days without any apparent effect. On February 8, 1912, he was presented before the Pediatric Section of the New York Academy of Medicine. Dr. Brill also saw

him at that time and made a positive diagnosis of Gaucher's disease. Shortly after this I noted the enlarged spleen in the younger sister and the diagnosis seemed more probable. On August 1, 1912, I made the following notes: The patient has a slight hemorrhage from the nose nearly every day when he sneezes or blows his nose. Anemia more marked. Some discoloration of the face, especially around the nose, of a yellowish-brown hue. Thickening of the conjunctivæ of a yellowish color on either side of the cornea. The spleen fills the greater

Fig. 1.—Author's patient showing outline of the spleen.

part of the left half of the abdomen and reaches 2 cm. beyond the median line. The liver 5 cm. below the free border of the ribs in the right mammary line. The axillary and inguinal nodes are palpable but not large. A venous hum is heard in the vessels of the neck, but no systolic murmur at the base of the heart. Wassermann negative; von Pirquet negative.

On Oct. 17, 1913, he was again admitted to my service. During the preceding two weeks he had had several severe hemorrhages from the nose and gums and was very pale. On one occasion he lost 6 ounces

of blood. He complained of feeling badly, had lost weight and was troubled with headache, dizziness, dyspnea and nausea. He was so distinctly worse that I decided to have a splenectomy performed. This was done by Dr. Henry Roth on October 22. The patient made a good recovery and was discharged Dec. 11, 1913. He then spent four weeks in a convalescent home in the country, gained nearly 10 pounds in weight and improved greatly. He still complains of occasional pains in the abdomen and legs. It is interesting to note the changes which took place in the blood following the removal of the spleen:

Date.	Hemoglobin Per cent.	Red Blood Cells.	White Blood Cells.	Poly-nuclears Per cent	Lympho-cytes Per Cent.	Eosino-phils Per cent.	
Oct. 18.	45	3,420,000	5,800	47	52	1	
" 21.	45	3,900,000	4,000	48	50	2	
" 22.							
OPERATION.							
Oct. 28.	55	3,140,000	15,000	71	25	3	
Nov. 3.	55	3,200,000	10,000	75	21	4	
Dec. 3.	45	4,200,000	22,800	65	35		Otitis (mild attack).
" 9.	52	3,300,000	15,000	62	35	3	

Of the 15 authenticated cases only 3 have occurred in males. Of these 3, 2 have come under my observation. These are also the only 2 males thus far operated upon.

To sum up the principal features, the patient presented the following manifestations: A disease beginning before the age of four and running a chronic course; a marked enlargement of the spleen and a moderate enlargement of the liver; no distinct enlargement of the lymph nodes; a yellowish brown discoloration of the face, especially about the nose and a wedge-shaped thickening of the conjunctivæ on either side of the cornea; a persistent leukopenia and a tendency to hemorrhages, especially from the nose. The size of the liver and spleen did not change materially during the seven years that the patient was under observation. As the disease progressed the blood showed a marked decrease in the percentage of hemoglobin (from 75 to 45), a less marked decrease in the number of red blood cells (4,390,000 to 3,400,000), a constant leukopenia with a gradual decrease in the percentage of polymorphonuclear leukocytes (70 to 48). As a result of the removal of the spleen there was an increase in the percentage of hemoglobin and an increase in the number of red blood cells and a much more marked

increase in the number of white blood cells (from 4,400 to 15,000), with an increase in the percentage of polynuclear cells (49 to 75).

It is too early to say that the patient is cured. That he has been very much benefited by the operation there can be no doubt. The spleen, although most markedly, is not the only organ affected, but it does not seem likely that an organ so distinctly changed can be of any value, and it may be that by removing it the body may be better able to cope with the as yet unknown toxic agent causing this disease.

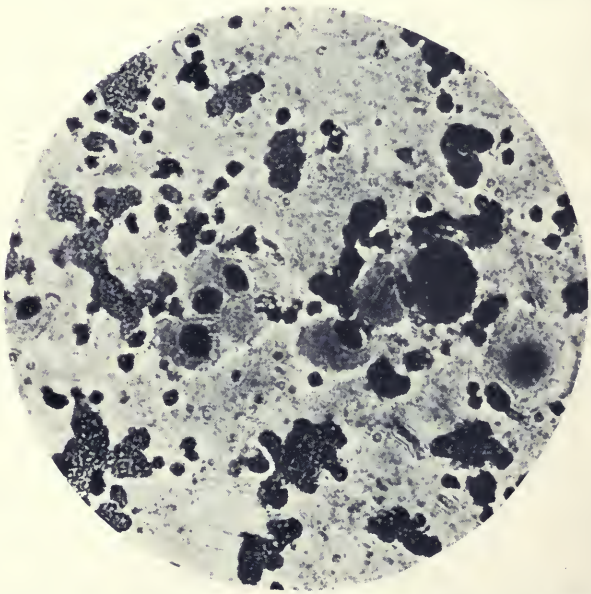


Fig. 2.—Smear from spleen pulp showing large cells with multiple nuclei, erythrocytes in masses and rouleaux. Few lymphocytes.

In a recent paper Dr. Niemann[1] reports a case of what is undoubtedly one of Gaucher's disease in an infant seventeen months of age from Czerny's clinic in Berlin. Curiously enough, his paper is entitled *"Ein Unbekanntes Krankheitsbild"* (An Unknown Disease or Symptom Complex). The clinical findings and pathological changes are those of Gaucher's disease, and the author himself thinks this diagnosis highly probable. The unusual features of the case are the early age at which the first symptoms were noted and the rapid course, ending in death. Cases have been already reported in young children and the disease does not necessarily occur in more than one member of the family.

The author proposes the name "grosszellige drüsen metamorphose" (large-cell glandular metamorphosis). The term "splenomegaly" is discarded because the disease is by no means confined to the spleen, and he prefers the term metamorphosis to hyperplasia because the pathological changes are more striking than is the increase in size. However, it seems likely that the term Gaucher's disease will be the one commonly used. Another interesting item in Niemann's case was the presence of a positive Wassermann reaction, notwithstanding which

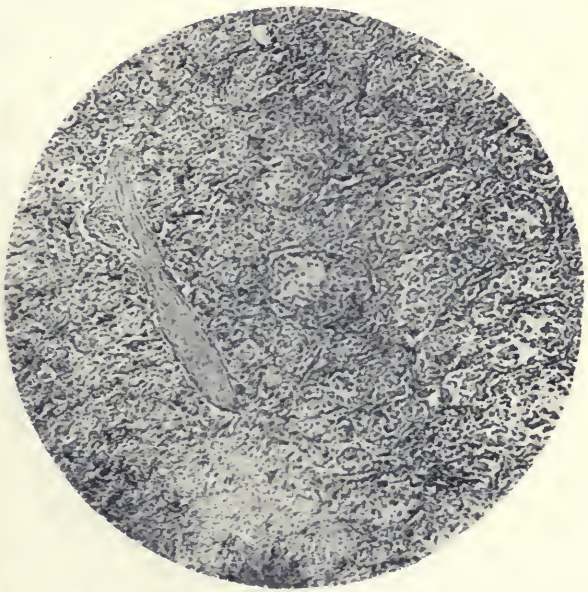


Fig. 3.—Low power of section of spleen showing venous sinuses.

antisyphilitic treatment had no effect and the post-mortem examination showed an entire absence of any syphilitic changes. The case proves that a positive Wassermann reaction is not absolute proof of the presence of congenital syphilis.

Now that a series of papers recently published have called attention to this rather rare disease, I have no doubt that a number of new cases will be reported within the next few years.

SURGICAL REPORT

On Oct. 22, 1913, under nitrous oxid gas and ether anesthesia, a vertical incision was made through the outer border of the left rectus muscle, sufficiently long to permit safe manipulation and delivery of the

enlarged spleen. Adhesions were present on the upper and posterior surfaces, which were readily divided after the vascular pedicle was first secured with a rubber covered clamp, ligated in sections with catgut and divided between ligatures. An attempt was made to separate the tail of the pancreas from the pedicle, but owing to some bleeding this was abandoned and the distal one inch of the pancreas was included in one of the ligatures and part of it was removed with the spleen. After covering the raw surface with peritoneum, a small cigarette drain was

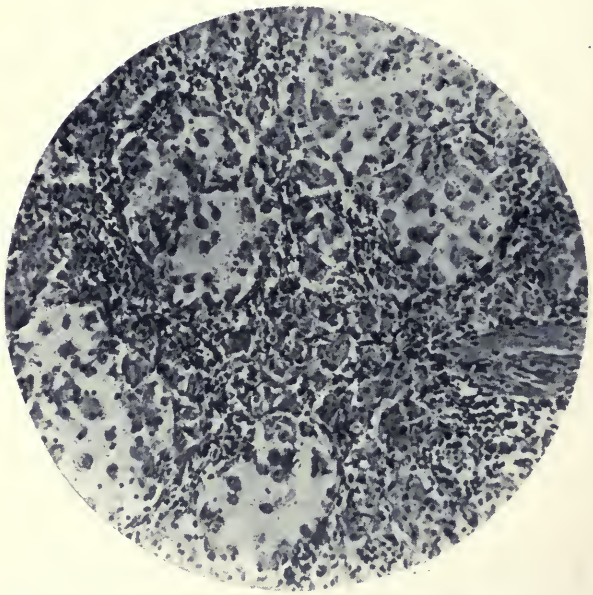


Fig. 4.—High power showing sinuses distended with multi-nucleated endothelioid cells and erythrocytes.

placed down to the stump. The abdominal incision was closed by several layer sutures. The patient stood the operation remarkably well. There being no discharge from the wound, the drain was removed on the third day and the wound healed by primary union. He was discharged from the hospital forty-nine days after the operation.

In a recently-published article on large-cell splenomegaly (Gaucher's disease), N. E. Brill and F. S. Mandelbaum[8] state that of the 14 authenticated cases of this disease, 8 had splenectomy performed, with 5 recoveries and 3 deaths as the result of the operation.

J. F. Erdmann and J. J. Moorhead[5] give a list of 10 operative cases, including 2 of Wilson's cases, which Mandelbaum, however, does not consider to be types of the disease under consideration. Furthermore, the one authenticated case of Gaucher's disease (G8869), reported by Wilson and operated by Wm. J. Mayo on Jan. 7, 1907, died. Erdmann and Moorhead are in error when they count this case as a recovery. A corrected list shows only 8 operative cases, with 5 recoveries and 3 deaths.

The accompanying table gives a list of cases submitted to operation:

Priority of Publication	Author and Date of Publication	Sex and Age	Blood Picture	Operator and Date of Operation	Weight and Size	Outcome	Duration of Enlargement	Other cases in Family	Priority of Operation
1	Picou and Ramond 1. 1896	Female 32 yrs.	Simple anemia	? 1895	2,800 gms.	R.	4 years	?	1
2	Bovaird 2. 1900	Female 16 yrs.	Simple anemia	A. J. McCosh May 17	12½ pounds	Died in 3 hrs.	13 years	Yes	2
3	v. Herczel 1907	Female 37 yrs.	Simple anemia	E. v. Herczel Sept. 27, 1905	3 kg. 32x16x12 cm.	R.	4 years	Yes	3
4	de Jong and vonHeukelom	Female 12½ yrs.	Simple anemia	von Rossem March 1, 1912	1,850 gms. 26x15x7 cm.	R.	6½ years	Yes	5
5	Mandlebaum 1912	Male 4½ yrs.	Simple anemia	J. Wiener March 1, 1912	490 gms. 18x9.5x5 cm.	Died next day	1 year	Yes	6
6	Wilson #G. 8869 1913	Female 27 yrs.	Simple anemia	W. J. Mayo Jan. 7, 1907	30x20x8 cm. 5,289 gms.	Died	15 years	?	4
7	Downes 1913	Female 28 yrs.	Simple anemia	Wm. A. Downes Dec. 14, 1912	1,813 gms. 35x13.5x6.5 cm.	R.	15 years	No	8
8	Erdmann and Moorhead 1913	Female 3¼ yrs.	Simple anemia	J. F. Erdmann June 6, 1912	430 gms. 17x10.5x7 cm.	R.	2 years	Yes	7
9	Herrman 1914	Male 13 yrs.	Simple anemia	Henry Roth Oct. 22, 1913	1,320 gms. 27x13x10 cm.	R.	10 years	Yes	9

SUMMARY

Female, 7. Male, 2. Recovered, 6. Died, 3.

Oldest, thirty-seven years; youngest, three years. Other cases in family, 6.

4. Niemann: Jahrb. f. Kinderh., January, 1914.

5. Erdmann, J. F., and Moorhead, J. J.: Am. Jour. Med. Sc., 1914, cxlvii, 312.

Longest duration, fifteen years; shortest, one year.

The patient presented by Dr. Charles Herrman is, therefore, the ninth operative case and the sixth recovery after splenectomy for Gaucher's disease. It is the third male case on record and the second male case subjected to splenectomy. It is the sixth patient operated upon in this country.

In view of the fact that all other forms of treatment have thus far failed to bring about the same amount of improvement which follows splenectomy, the latter procedure seems justifiable because, even if the improvement proves to be only temporary, splenectomy will surely relieve the symptoms caused by the increased size and weight of the spleen. Furthermore, the improvement in the condition of the blood tends to indicate that the removal of the spleen in this disease has a beneficial effect. Splenectomy is a comparatively safe operation, providing it is done before the general condition of the patient becomes such that any other procedure of such magnitude is too dangerous.

PATHOLOGICAL REPORT

The spleen in this case measured 27 x 13 x 10 cm. in its various dimensions and weighed 1,320 grams (almost 3 pounds).

The surface was wrinkled and of a translucent purple color.

On cut section the capsule is seen to be somewhat thickened. The parenchyma is of a brownish-red color, mottled everywhere with minute hemorrhagic areas about the size of a pin-head. The organ cuts with considerable resistance, nowhere exuding blood, and presents a firm homogeneous surface from which very little pulp can be scraped off. Malpighian bodies are not distinguishable.

While the microscopic picture of this disease is absolutely diagnostic, yet the interpretation of the findings is still in doubt.

In spreads made from the splenic pulp immediately after operation, fixed in methyl alcohol, and stained in Giemsa's solution for twenty-four hours, we see numerous large endothelioid cells, with irregular outlines, containing one or more fairly large, round or ovoid shaped nuclei, which are rich in chromatin and are usually situated eccentrically. The number of nuclei in one cell may be very large—in this case the highest counted being seven. The protoplasm of the cell is roughly homogeneous. Phagocytic cells could not be found. (Fig. 1.)

On microscopic section the most prominent feature is the enormous distention of the venous sinuses, which are filled with erythrocytes and

the above described endothelioid cells. The sinuses are round or oval in shape and lined with endothelium. Some sinuses, however, appear to be empty except for a few cells scattered along the endothelial wall. Here and there are found islands of fairly normal pulp. (Fig. 2.)

The wall of the sinuses is composed of connective tissue supporting the endothelial cells, but reticulum fibers are never found extending into the lumen of the alveoli.

This is the typical pathological picture of the spleen in this disese, which still remains to be explained.

The American pathologists are inclined to favor the theory that these large cells arise from the endothelium lining the sinuses, while the Germans claim that they come from the reticulum. As no transition stage from either source has been seen, both theories are unproved. Still others suggest their derivation from the lymphocytes—also without substantiation.

The possibility of a protozoon as the etiological factor has been advanced, but all cultural and animal experiments to prove this have given negative results.

In this case cultures were made aerobically and anaerobically after the method of Noguchi, but without result.

The only point of record among all authors is that these cells must arise from the hemopoietic system.

Besides being found in the spleen they are also present in the liver, lymph nodes and bone marrow.

DISCUSSION

DR. SOUTHWORTH: Did Dr. Knox consider recommending operation in the younger child of the same family as a preventive measure, with the idea of seeing what could be done toward limiting the progress of the disease?

DR. HOLT: I would be glad if the author would give results in other cases reported in literature, in which the patients have been operated on.

DR. KNOX: I should like simply to point out, as Dr. Herrman has already said, that here we have now one clear-cut group in the cases of splenomegaly which can be separated from the others. Whether the diagnosis can be made more than tentatively, without pathologic examination, I think is a little questionable. When, however, more than one member of the same generation in the family have similar symptoms, the diagnosis is exceedingly probable.

I agree with Dr. Herrman that the time to do splenectomy is early; when the lesions have extended to other organs, as they did in my case—the liver and lymph glands being involved—it seems hardly probable that splenectomy will help. We have had no reports of more than a short time after this operation. It may be possible with a large spleen of the kind that we are

considering, or a liver close down to the wall of the abdomen, to needle the spleen or liver and to extract some material from which a positive diagnosis could be made.

DR. HERRMAN: In reply to the question as to whether I advised operation on the sister of the patient, that she was in comparatively good condition and it would therefore be difficult to obtain the parents' consent until the symptoms became more marked. I suggested a puncture of the spleen for diagnostic purposes. In this way a little of the spleen pulp could be obtained and probably a number of the characteristic cells would be found. This is the only way of making a positive diagnosis before operation or necropsy. The surgeon whom I consulted thought the procedure dangerous so that it was not done. As to the patients operated on, including the one just reported, there were nine in all. The first one operated on in this country was a patient of Dr. Bovaird's, operated on by Dr. McCosh in 1900. Of the nine cases reported, seven patients were women and two men; six recovered and three died; the oldest patient was 37 years of age, and the youngest 3 years; in six of the cases other members of the family were affected. I believe that in the future more of these patients will be operated on. The operation itself is not attended with any great difficulty or danger; the spleen has frequently been removed successfully in cases of rupture and for other conditions. It is the only method of treatment that gives any definite result; the treatment with drugs and the Roentgen ray had no appreciable effect. Most of the patients operated on have been followed for too short a time to judge of the ultimate result, but most of them have been decidedly improved.

SHALL A DEPARTMENT OF PEDIATRICS INCLUDE IN ITS CURRICULUM THE THEORETICAL AND PRACTICAL TRAINING OF MEDICAL STUDENTS IN THE SOCIAL-MEDICAL WORK AMONGST INFANTS AND CHILDREN?

H. J. GERSTENBERGER, M.D.
CLEVELAND

Had the 1913 copy of the Transactions of this Society reached me before I had expressed my willingness to the president to present this paper to you at this annual meeting, I should not have chosen this subject, for the simple reason that at your last meeting practically all that I had in mind was said in the most clear and excellent manner by Dr. J. C. Gittings[1] in his paper on "The Relation of the Physician to Social Service."

Dr. Gittings had expressed better than I personally could the ideas that I have had and have to a big degree carried out since 1905 in the development of the Babies' Dispensary at Cleveland, regarding the qualification and training for, and regarding the position of the physician and nurse in social service work, and finally regarding the conception of social service work.

Therefore, I need not repeat what he has told you, but can pass directly to the question, shall a department of pediatrics include in its curriculum the theoretical and practical training of medical students in the social-medical work amongst infants and children?

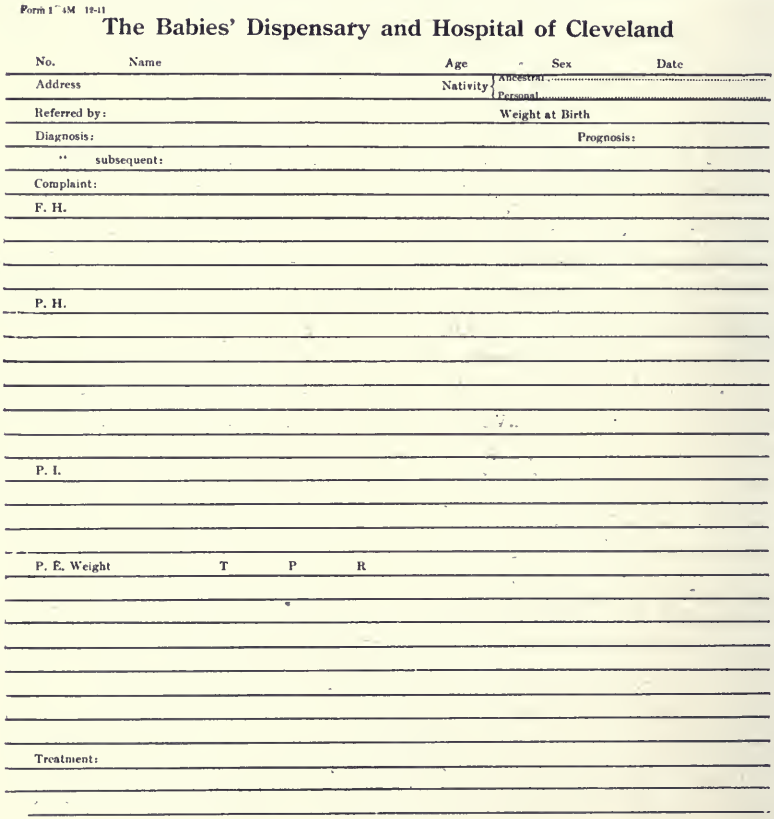
I believe that all of us here agree with Dr. Gittings' statement that "between prevention of disease and preservation of health there is no essential difference, and surely the physician and not the sociologist should become the logical arbiter of all problems relating to health."

If we are willing to take this first step, then we must be ready to take the second, namely, to see to it that "the logical arbiter" is adequately trained and prepared for his work, especially when we are aware that the future welfare and development of the medical

1. Gittings, J. C.: The Relation of the Physician to Social Service, Trans. Am. Ped. Soc., 1913, p. 101.

profession and its importance in the eyes of the public at large depend more on the manner and thoroughness in which medical men are trained in this work than on any other one factor.

The most difficult part, however, in carrying out this idea is this: that the medical profession at large, together with the faculties of the medical schools, do not to-day realize that both for the prevention



Form 1—4M 12-11

The Babies' Dispensary and Hospital of Cleveland

No. Name Age Sex Date

Address Nativity { Ancestral / Personal

Referred by: Weight at Birth

Diagnosis: Prognosis:

" subsequent:

Complaint:

F. H.

P. H.

P. I.

P. E. Weight T P R

Treatment:

Fig. 1.—Physician's record.

of disease and the preservation of health, as well as for the welfare of the profession itself, it is essential that medical men adequately train themselves in this important field. Both the faculties and the practicing medical profession seem still to feel that social medicine is beneath their dignity, that social workers, laymen, and others had better apply their time to it rather than they. The sad fact to all of us who "see"

is that laymen and social workers in by far the greatest proportion agree with this stand of the present day medical profession and medical school faculties and, therefore, those of us who "see" have an especially difficult task in education to perform.

Whenever any medical work, especially that which deals in a practical manner with the public at large, gets away from a more

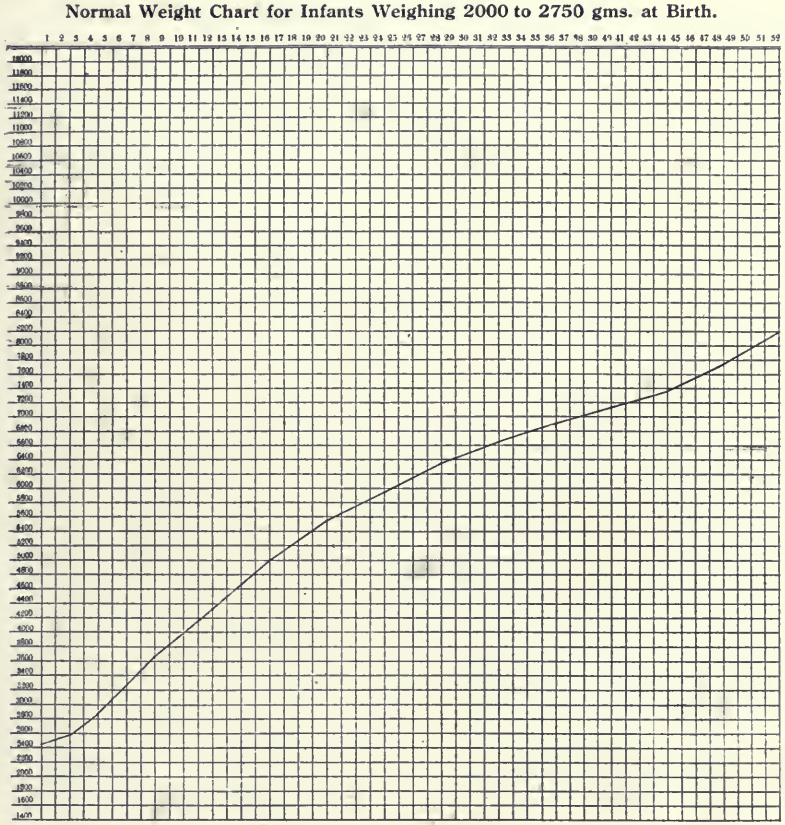


Fig. 2.—Weight chart, part of physician's record.

or less direct contact with the fountain of truth and progress, namely, from scientific medicine, then there is a grave danger to the present and future standing and effectiveness of the medical profession and to the safety of the public as well.

Dr. Thomas B. Cooley[2] very well stated that "social work is becoming so popular that there is some danger of its being attempted without skilled direction which is essential to success."

The first requisite for any work ought to be that it be built and operated on the solid foundation of the scientific facts of the day.

Form f 4m 4 15

NURSE'S REPORT

_____ Nurse

No. Name Rate Date

Address	No. Rooms	Rent	Taxes	Cleanliness	Light	Ventilation	Landlord

Name	Character	Occupation	Place of Occupation	Income	
				Total	Partial
Father					
Mother					
B. S.					
B. S.					
B. S.					
B. S.					
B. S.					

Other Sources of Income Amt of Income

Material Aid

Medical Aid

Material Circumstances

Milk Supply

Ice or Ice Box Available

Are Instructions Followed? (If not, Why?)

Church

Fig. 3.—Nurse's record.

If, as is most often the case to-day, especially when laymen and social workers direct the work, the cry and plan is, above all, cooperation—at the expense of the first-hand knowledge of the facts—then I for one wish to go on record as saying that I believe that such work

2. Cooley, Thos. B.: Relation of the Infant Welfare Movement to Pediatrics, Jour. Am. Med. Assn., 1912, lix, p. 2217.

even when based on as good a cooperation as can be made, is a sham, a waste of time, a synonym for superficiality, and cannot be compared with the result, although this result may never be announced with trumpets and bells, that will be obtained by work based on facts and truths even though it lack the desired cooperation.

orm 5st 1M 5-18

The Babies' Dispensary and Hospital of Cleveland
Boarding Home Supplement to Regular Nurse's Chart

Nurse (Date)

No. Name of child Age

Nationality: Ancestral Persoonal: Religion

Referred by: (Date)

Detailed statement as to why child is to be boarded:

Name of father and mother:

Address of father and mother: (Date)

Are there any evidences of tuberculosis, syphilis or gonorrhoea in the child or in any family member or individual who has cared for child?

(a) from your own observation ; if so (in whom; when and what)

(b) from observation ; if so (in whom; when and what)

Diagnosis (original) of child (Date and by whom):

Diagnosis (subsequent) of child (Date and by whom):

Since boarding with B. H. Chart No.

At expense of:

For further information see Nurse's notes (Date)

Since boarding with B. H. Chart No.

At expense of:

For further information see Nurse's notes (Date)

Fig. 4.—Boarding Home record—for boarder.

How much good do we do by giving excessive amounts of the best milk, certified, if you please, to anemic children when most of them will be benefited by a reduction and not by an increase in the quantity of milk?

How much useless expenditure of money has resulted from the overfeeding of tuberculous patients with eggs and milk by associated charity organizations, church societies, nurses, and even by physicians who have gotten out of touch with the progress in medicine or one of its branches?

Form 59 1M 5-18

The Babies' Dispensary and Hospital of Cleveland

Boarding Home Chart

Nurse (Date):

No. Name of Foster Mother: Age S. M. W. D.

Referred by: Religion: Wet Nurse

Born in In U. S. Since Character: Disposition:

Date	Address	Tel.	No. Rooms	Rent / Taxes	Cleanliness / Light / Ventilation	Porch / Roof / Yard	T.B. Record	Landlord

Date	Husband's Name	Character / Disposition	Occupation	Place of Occupation	Present Abode

Number Adults in Home Ages No. Children in Home: Ages

Tubercular in Home: Syphilitic in Home: Epileptic in Home:

Reasons for taking Boarders.

Previous Boarders (for whom and when):

Ability to prepare Food at Home (Date):

Cooling Facilities (Date):

Possible Milk Supply (Date):

Total Family Income (Date):

Total Family Debt (Date):

Material Circumstances (Date):

General Impression of Boarding Home (Date):

Contract Signed (Date).

Fig. 5.—Boarding Home record—for foster mother.

The method that undoubtedly will bring the best results is the one that combines in itself, first, the solid foundation of scientific facts; second, the practical training that permits the individual to best apply these facts in the cure, and especially in the prevention of sickness and death; and third, the cooperation that brings harmony in the work and lessens wasteful reduplication.

There is no field in medicine to-day which gives greater opportunity for the successful application of preventive medicine than that which deals with infancy and childhood. Not only do the infant and young child point more clearly and readily to the various dangers that beset mankind than do adults, but they at the same time give us the oppor-

Name of Boarder: Chart No. at Central and Branch Dispensary:

Referred by (Date): Medical Supervision:

Amount to be paid for Boarding per (with-without milk) by:

Date of Acceptance of Boarder: Of Dismissal:

Result (a) Child:

(b) Foster Mother:

Special Remarks:

Name of Boarder: Chart No. at Central and Branch Dispensary

Referred by (Date): Medical Supervision:

Amount to be paid for Boarding per (with-without milk) by:

Date of Acceptance of Boarder: Of Dismissal:

Result (a) Child:

(b) Foster Mother:

Special Remarks:

Name of Boarder: Chart No. at Central and Branch Dispensary:

Referred by (Date): Medical Supervision:

Amount to be paid for Boarding per (with-without milk) by:

Date of Acceptance of Boarder: Of Dismissal:

Result (a) Child:

(b) Foster Mother:

Special Remarks:

Fig. 6.—Reverse side of Boarding Home record for foster mother.

tunity to improve the strength and health of the future nation by prevention.

There is every reason to believe that when we through proper nourishment, housing and care save the body and mind of infants and children from as many as possible of the various types of injuries that may be inflicted on them, that we are saving their resistance,

increasing their tolerance, and giving them a better chance of becoming the healthy future men and women of the nation.

The widest application of preventive medicine will sooner or later be accomplished through the state and especially through the municipal health departments—the centers of the practical work. In these

City of Cleveland

DEPARTMENT OF HEALTH—BUREAU OF CHILD HYGIENE—BLIND RECORD No. 1

Nurse

No. Name Age Sex Date

Address

Nativity. Ancestral Personal

Referred by at M. 191

Name of Father of Mother

Name and address of midwife at confinement

Name and address of physician at confinement

Date and hour of birth Has birth been reported to health office?

Date and hour of beginning of eye symptoms Of treatment by physician

Date and hour of report to health office To physician

Name and address of treating physician

Kind of treatment instituted By whom?

Why was no physician called to treat eyes?

Number of adults in home Number children

Name and age of those having or have had eye disease (kind)

Number rooms Cleanliness Light Ventilation

Material and medical relief

Total weekly income; actual Maximum

Material circumstances

Church Landlord

Diagnosis

Diagnosis microscopic (date and by whom)

Nourishment of infant

If not on breast, state why

Treatment (date and hour of beginning; kind; under whose direction)

Fig. 7.—Record for blind.

health departments the division of child hygiene will be the most important and the most far reaching. Around this division, if around any division of the health department, the remainder of the public health work must be built. I mention this here because of the fact that those who have been attacking the tuberculosis problem have

finally come to realize that "next to the prevention of the spread of tuberculosis bacilli by proper isolation of all open cases of tuberculosis, the work carried out by the prophylactic babies' and children's dispensaries to prevent illness and death amongst infants and children does more than any other one activity toward the prevention of tuberculosis, because it uses, or at least aims to use, all of the means at its disposal to attack every factor that is instrumental in lowering the resistance of the infant and young child toward all diseases and harmful environmental conditions." For this reason and for the additional one that they are the oldest and the most widely organized of the social-medical organizations throughout the country—in cities this in many instances is not so—they are hoping to group all the other branches of public health work around the tuberculosis department. I argue that the modern pediatrist will be better able to direct the practical work of the reduction of tuberculosis than a specialist for tuberculosis will be able to direct the practical reduction of sickness and death amongst infants and children. The latter's activity is directed toward the elimination of the one disease, while the former is fighting all of the harmful influences that may hamper the welfare of the infant and child. In other words, he must be in touch, if possible, with all of the progress that is being made in the prevention and treatment of all of the medical and social ills that beset the infant and child. I do not feel, however, that even this is the ideal plan, but am simply pointing out that to center all public health work around a department or an association for prevention and reduction of tuberculosis would be a step backward instead of forward.

The ideal scheme for the practical application of preventive medicine in public health work would seem to me to be the one which plans to place one dispensary in a small district with a well trained and well paid full-time physician in charge, aided by an adequate number of well-trained nurses for nursing and social work and one or two medical assistants, if necessary, to care for all of the medical and social ills of the people in his district, except for the obstetrical and post-partum care of the mothers. This physician would have as his administrative chief the commissioner of health, and as his vigilant advisers and counselors for the different parts of his own work, men at home in the special fields of medicine. I should say that in America these consulting advisers should be the heads of the

respective departments or sub-departments of the university medical school, or some one of the university staff appointed by such a head, for the simple reason that after all is said and done our greatest hope for completeness of knowledge, for ideals and for stable policies in medical education in America are the universities. This would mean in the case of child hygiene work that the professor of pediatrics would be, in all probability, through a member of his staff, the adviser to the men in the district who are attempting to make practical application of preventive medicine. In this manner it will be possible to see that the men in the district are given the benefit of the most recent accomplishments in pediatrics; and on the other hand, it will enable the university head to keep informed with what degree of success these new facts can be applied in the field of public health. The important point is that a tie is established between the medical school and the health department which will be of benefit to both parties, but especially to the third—the public.

I am sure that we pediatrists, and especially those who have charge of departments, have a big duty to perform in at least opening the eyes of the future medical men—the medical students—to this vast and important field of activity and the manner in which it ought to be tilled. By doing this we will at the same time more effectively than in any other manner get the faculties of the medical schools, the practicing medical profession, and the public at large, to realize that pediatrics is nothing more or less than the broadest type of internal medicine applied to the most sensitive and most easily influenced group of human beings—the infants and children—and that therefore, it is second to no other department in its opportunity for valuable work and results, both scientific and practical, preventive and curative, and that it, therefore, deserves in the councils of the medical faculties the same recognition as is given to medicine, to surgery, to obstetrics, and to the so-called laboratory departments.

As I have stated above, I am convinced that the most important and far reaching department of public health work is that which refers to the protection and care of infants and children, and feel that this in itself is an adequate reason why a department of pediatrics should consider it its special duty to open the eyes of the medical students to this new field for medical men. It should go about this by giving to the men above all the best possible training in the fundamentals of

scientific pediatrics, by augmenting this by practical milk laboratory work, and finally by showing them by personal contact with the child, the home, the family, the surroundings, the dispensary, and various social, religious, philanthropic and other organizations of the community, how to apply scientific pediatrics in actual practice, with the idea to prevent and cure.

This has been the plan that we have followed in Cleveland since 1911. During the junior year all of the time is spent in teaching pediatrics in the form of clinics and lectures; during the senior years the class is divided into four sections and each section spends one and one-half to two hours per week for eight consecutive weeks at: (a) ward walks in the contagious ward of City Hospital; (b) ward walks in the babies' and children's ward of Lakeside Hospital; (c) practical milk work in the milk laboratory of the Babies' Dispensary; and (d) practical work in the prevention of sickness and death amongst infants and children by visits to the prophylactic dispensaries and to the home, by contact with the infant in the prophylactic dispensary and in the home, with the family, the surroundings, the ideals and methods of operation of various charitable, philanthropic, religious and municipal agencies that may be used in improving the condition of the infant and the entire family.

We have found that this work—(c) and (d)—is appreciated by the men—at the beginning not as much as at the end—and we are convinced that the seed has been sown from which will grow the plant of knowledge that all public health work and social-medical work must be based on scientific medicine, but that this, without the knowledge of how to apply it in every day life, means little, and that, therefore, the ideal plan is the one which embodies, first, the solid foundation of scientific facts; second, the practical training that permits the individual to best apply these facts in the cure, and especially in the prevention of sickness and death; and third, the cooperation that brings harmony in the work and lessens wasteful reduplication.

N. B. Copies of the "Medical," "Nursing," "Blind," "Boarding Home" (one for boarder and one for foster-mother) records are given below and best present the points to which the students' attention is called.

(A) PROPHYLACTIC BABY WORK, INCLUDING CONSIDERATION OF MEDICAL AND SOCIAL FACTORS

UNDER DIRECTION OF DR. C. W. WYCKOFF, INSTRUCTOR IN PEDIATRICS, WESTERN RESERVE MEDICAL SCHOOL

Given in Board of Health Prophylactic Babies' Dispensaries and out in the districts, Thursdays, a. m., 10:30 to 12:00, for eight consecutive sessions, to one-quarter of senior class in pediatrics of Western Reserve Medical School, *during 1911-12, 1912-13.*

I. General outline of Babies' Dispensary Prophylactic Work at Babies' Dispensary
   a). Infant mortality
   b). Milk production

II. Prophylactic Babies' Dispensary of Board of Health. Outline Practice. (History taking, weighing.)

III. General hygiene of infant
   1. Daily bath
   2. Feeding: Breast, artificial food
   3. Clothing, sleeping, airing

IV. Prophylactic work in home
   a). Social history
   b). Teaching mother
      1. Care of breasts
      2. Preparation of artificial food
      3. Bathing, clothing, sleeping, airing
      4. Mother's own state of health

V. Blind work
   a). Municipal
   b). State

VI. Boarding Home: Need of; demonstration

VII. Public school infant hygiene

VIII. Associated Charities, Humane Society, Juvenile Court, Orphan Asylum, Maternity Dispensaries, etc.

(B) PROPHYLACTIC BABY WORK, INCLUDING CONSIDERATION OF MEDICAL AND SOCIAL FACTORS

UNDER DIRECTION OF DR. C. W. WYCKOFF, INSTRUCTOR IN PEDIATRICS, WESTERN RESERVE MEDICAL SCHOOL

Given in Board of Health Prophylactic Babies' Dispensaries and out in the districts, Thursdays, a. m., 10:30 to 12:00, for eight consecutive sessions, to one-quarter of senior class in pediatrics of Western Reserve Medical School, *during 1913-14.*

I. Dispensary clinic one and one-half hours. Each student is given a baby to examine and watch prophylactically
   Fifteen minute talks at end of period on feeding, bathing, clothing, and general physical and mental development of infant. Also on housing, financial and social conditions of family

II. Same as No. 1

III. Same as No. 1

IV. Same as No. 1

V. District: "Dirty home" demonstration—three students going to a home under supervision of an experienced social service nurse. Home is thoroughly examined according to printed directions
A home milk modification is made
VI. District: "Clean home" demonstration—three students going to a home under supervision of an experienced social service nurse. Home is thoroughly examined according to printed directions
VII. Bath and clothing and bed demonstration at Central Dispensary
VIII. Talk on and demonstration of work carried out to prevent blindness in the new-born
All the work of the medicosocial agencies affecting infants and children is summed up

(C) PRACTICAL WORK IN THE PREPARATION OF ARTIFICIAL INFANT FOODS

UNDER DIRECTION OF DR. C. W. WYCKOFF, INSTRUCTOR IN PEDIATRICS, WESTERN RESERVE MEDICAL SCHOOL

Given in the Milk Laboratory of the Babies' Dispensary, 2500 East Thirty-Fifth Street, on Mondays from 4:30 to 5:30 p. m., for eight consecutive sessions, to one-quarter of senior class in pediatrics of Western Reserve Medical School, *during 1911-1912, 1912-13, 1913-14.*

I. .General introduction into the central milk laboratory of the Babies' Dispensary
a). Care and management of certified milk from cow to infant, i. e., pasteurization, boiling, cooling, transportation, etc.
b). Care of bottles, nipples; kind, number
II. Simple milk dilution. Sugars: kind, value, weight, measure, per cent. for different infants (age, weight, condition)
III. Preparation of tea, oatmeal, barley, rice and albumin-water
IV. Preparation of skimmed milk, top milk, farina soup. Fat test
V. Preparation of vegetables
VI. Preparation of Keller's malt-soup. Indication for using
VII. Preparation of buttermilk. Indication for using
VIII. Preparation of Eiweissmilch. Indication for using

DISCUSSION

DR. CHAPIN: I think that hospital and institutional statistics, as such, are of little value unless they take into account the ultimate condition of the child, and it seems to me that this question of Dr. Gerstenberger's is most timely and valuable. We all know that institutional work is very different from home work. Many nurses who do splendidly in the hospital are failures in the home, and many physicians too, interns, who make good records in the hospital seem to be utterly unable to adjust themselves to the varied conditions of life in the home, particularly in the poor home. I think if the universities could give time to this instruction it would be the most valuable thing in actual results, so far as these children are concerned.

The only trouble that occurs to me is that the universities will not give the time. They do not give time enough to scientific pediatrics as it is. Many men who are turned out every year are poorly trained, as we see them now. That is not the fault of the professors; it is the fault of the university authori-

ties, who put pediatrics on the same basis as the most minute specialties. They give very little, if any more time to this, the most important branch of internal medicine, than perhaps to diseases of the ear. I think that is the most crying evil of the day: men turned out insufficiently trained. I think a society of this sort could use its influence there, as well as in the broader field that Dr. Gerstenberger has mapped out.

I may say, with reference to the actual social service, so far as I know, the first attempt was made at the children's ward of the New York Post-Graduate Hospital; that is, in 1900 social service was begun there and has continued ever since. Perhaps I am mistaken; but I think that was the earliest date that hospital social service was ever started. Since that date every home has been regularly visited and the report of the life conditions of the children is turned in, so that the attending physician can know, so far as possible, the conditions surrounding that child in the home and the disadvantages that it is living under. I believe that in the future this work is going to loom up greater and greater; and I thoroughly believe, with Dr. Gerstenberger, that the doctor—the scientific man—should be the one to oversee it. We are in danger of the kindly people, the sentimental people, the untrained people who rush into this work. It should be the physician that should oversee it.

It may be of interest to know that we first started with women physicians in this work: for quite a number of years we employed a woman physician; but I do not know why. They did not do as good work as trained nurses in this particular line, so that for the last seven or eight years we have had a trained nurse do the work instead of a woman physician; although we would suppose that the physician would do it better; but it requires a peculiar personality for this work. A woman does it better than a man; but the woman must be broad gauge—she must have an enthusiasm for humanity in her heart; she must be no technician; she must have a tenderness for these suffering children; and, strange to say, that is not always easy to get. I sometimes think it is easier to get among men than among women of certain types.

DR. BLACKADER: I only want to ask one or two things. I have listened with a great deal of pleasure to Dr. Gerstenberger's address; it is, to a certain extent, ideal; but whether it is an ideal we can reach in teaching school is another question. I would like to know how many years he has his students under his control.

DR. HOLT: I do not think the outlook is quite so bad as pictured. I think pediatrics is given very much more time than formerly. Dr. Chapin's statement that pediatrics in many of the medical schools gets no more time than diseases of the ear certainly does not hold at Columbia. Here pediatrics gets as much attention as obstetrics, and I think it gets more hours. Generally speaking, of course there are exceptions, but the proper recognition of pediatrics is becoming more and more evident all over the country. We know how it has been at Harvard for many years; we know how it is coming to be at Johns Hopkins, and now at Cleveland and at St. Louis. I think there are many signs of encouragement. I think that the men turned out from our best medical schools have a pretty fair knowledge of pediatrics. We have now at Columbia in the curriculum about 130 to 140 hours devoted to pediatrics. That work is not optional, it is work required for the degree in medicine. We can hold up a man for graduation if he does not pass his pediatric examination.

I heartily agree with what Dr. Gerstenberger has said about the relation of the physician to social work. Those of us who attend the meetings of the

Society for the Study and Prevention of Infant Mortality have been really chagrined to see how the physicians, and especially the pediatrists, have rather held aloof from that work, and how it has so largely drifted into the hands of the social workers. I think those of us who are really in sympathy with this work ought to indicate it by our presence and our support. In the meetings of this society are brought together the workers in this line from all over the United States; measures are discussed and methods are considered. If we criticize other workers we must blame ourselves for not doing our own share in directing this work. Dr. Gerstenberger is quite right in saying that the medical student must have his introduction to this work. We must not wait until after graduation. When the doctor is out in practice, too often he has too many other things to do. I believe that the future is going to see a greater demand on the part of medical men everywhere for knowledge of preventive medicine and public hygiene. Perhaps this will be the most important part of our work. The medical student must be trained, in some measure at least, to enter this field of activity.

DR. TALBOT: The next meeting of the Society for the Prevention of Infant Mortality will be held in Boston and we hope that any members here who are not members of that society will join, because we need members very much. We have in the Massachusetts General Hospital a very well-regulated social-service department; and we are continually saying that we could not get along without the social-service workers. We have found that the social-service department there is continually trying to get us to take the responsibility of the work, and the criticisms that I have heard of social-service work have been criticisms from the places where the medical men would not take the responsibility and left it to the enthusiasts who would like them to take it.

DR. CHAPIN: The point raised by Dr. Holt—that I had not attended any of those meetings—is true; and I would like to say why I have not attended, so long as the subject is up. It brings me back to the address of the president: that the work should have been done by this society, and not by the constant multiplication of other societies. I regard this as an evil.

DR. KERLEY: In my address at Delaware Water Gap (I had the honor of being president of the society) I suggested that this line of work be taken up and form a part of the utility of the society; it was supported by Dr. Rotch only. That was a very few years ago.

DR. KNOX: Dr. Gerstenberger's paper was interesting to me, as I have been engaged in this kind of work for two years and I want to endorse everything that he says.

In reply to Dr. Chapin, if we had waited for this society to start this work we would have lost by this time thousands of babies' lives. We are glad that the people who are interested in this work look still to the members of the society for leadership and urge them, as Dr. Talbot has said and as you have evidence in Dr. Holt's acceptance of the presidency of the American Association for the Study and Prevention of Infant Mortality last year, to take the leadership in the direction of the work; but it can not be done entirely by physicians. A large part of the prophylactic work must be done, it seems to me, in the first place by mothers and next by the social workers and by anybody who is fond of the baby, and a large majority, I may say, of these people are not found among physicians. Very many of them are, but they are also engaged in various laboratory researches where they cannot do the actual work but can counsel those in the field. It is the cooperation of all elements of

the community fond of the baby, interested in saving its life, under the direction, it seems to me, of physicians—to such a combination we can look to accomplish our most enduring results.

Now, as regards the opportunities in the work of this kind for medical students. It seems to me they have been almost neglected in the past. We have in Baltimore consultations attended by from twenty to one hundred babies in the afternoon under the charge of a physician and nurses. What can be more useful as an opportunity for a student to learn how to prevent illness and death among babies than a service at such a consultation? This work is not merely curative; it is largely preventive. One does not wait until the baby is ill before his services are applied. He sees well babies and tries to prevent illness. What better preparation could there be for the highest function of a physician than to meet these scores of well babies coming in week after week with their mothers, and to advise how to keep them well throughout the entire year. How valuable the opportunity to see and treat the little illnesses that rarely are seen in medical clinics or hospitals and which will surely be met in private practice.

I think, too, that pupil nurses ought to be sent to these consultations where they can see these babies—well babies—and take part in this same campaign to keep them well. I believe the future will see the designation, in rotation, of the whole student body taking the course in pediatrics for service at these welfare stations, so that every student and every nurse who goes out from the medical schools will not only be interested in this great crusade for baby welfare, but will know how to keep babies well and not only how to treat their symptoms after they have become ill.

DR. MORSE: One of the last things that Dr. Rotch did in the organization of the Pediatric Department of the Harvard Medical School was to introduce a course on preventive pediatrics, given during the fourth year. Our fourth year, as you know, is elective; that is, the men elect what they will study in their fourth year; and they elect the courses for a month at a time. In each one of these months one afternoon a week was devoted to preventive pediatrics, a series of talks being given by different men on the prevention of the different types of disease. We found that this method was not satisfactory. We are planning next year, instead of giving that sort of a course, to put the fourth-year men into the Milk and Baby Hygiene Association stations for an afternoon a week during the month.

In the new School for Health Officers which has been established by Harvard University and the Massachusetts Institute of Technology together, I gave a course of lectures last winter on infant mortality. Dr. Howard also took the physicians to the Milk and Baby Hygiene Association Stations for two or three afternoons. Then in the last two summers, in our Summer School for Medicine Dr. Howard has had four exercises during each month, in which he has taken the graduates to the Milk and Baby Hygiene Association stations. He has also given them a series of talks on preventive pediatrics; that is, on the prevention of infant mortality.

The Children's Hospital in Boston has not taken the part in social service work in the past which it ought to have done. It has had a social-service department, but I regret to say not so effective a one as it ought to have had. We are trying to improve that as fast as we can. My observation in Boston is that there is not sufficient correlation—I hate that word, because it is used so much—between the different agencies which are engaged in the work of

looking after babies and children. A number of people in Boston are now at work there on a very large scheme to try to systematize the work of these different organizations.

DR. GITTINGS: From the present indications it is not inconceivable that in the future we will all be on the Chinese basis and be paid for preventing disease, not paid when the patient is sick! Seriously, I want to emphasize how strongly I feel with regard to Dr. Gerstenberger's position. The physician with a well-established practice can not be expected to do more than extend a hearty sympathy to the work. Plenty of workers are available. The necessity is for trained medical directors of the work, not workers. As soon as the idea is planted in the minds of the students it is only a question of time before a certain number of these men will qualify for this particular specialty.

DR. HAMILL: When Dr. Gerstenberger said that the president of the society had suggested this subject to him, he should have said that he suggested the subject to the president, rather hesitatingly, and the president urged him to present it and he feels especially grateful at present that he did so.

I want to say just one word, also, in answer to Dr. Chapin's criticism of the American Association for the Study and Prevention of Infant Mortality. It seems to me that the great advantage of the organization is that it gives us a point of contact with the sociologist that we cannot get effectually in any other way. The association is much in need of the help and influence that the membership of this society could give; and if Dr. Chapin and the other members would attend a few of its meetings I think they would very readily understand why it is that those of us who are members of the association and interested in its welfare are extremely anxious to have the support of the pediatric profession. Dr. Talbot has told you that the next annual meeting is to be held in Boston in November. Boston is in close proximity to most of the eastern cities; and I sincerely trust that as many of you as can will not only come to the meeting, but will become members of the association.

DR. CHAPIN: There is a meeting here; there is a meeting of the Milk Commission in Rochester; there is a meeting of the American Medical Association in Atlantic City. Now there is another meeting in Boston.

DR. HAMILL: That is in November.

DR. CHAPIN: That brings up the point I make—the great difficulty for the members to make a living who run all over the country—the necessity to concentrate!

DR. HAMILL: I think you would benefit the cause of humanity more by going to the meeting at Boston than by going to the meeting at Atlantic City.

DR. GERSTENBERGER: Judging from the remarks of Dr. Blackader, I am sure that he misunderstood me about certified milk. I did not speak against certified milk; I simply stated that even certified milk might be misused if men did not know how and when to use it.

Regarding the statement that it is unwise to add anything more to the teaching responsibilities of pediatric departments because the curriculums of the medical schools are already overfilled, I can only say that I think it is impossible to try to teach a student any subject from A to Z. I think what we as teachers must try to do is, first, to get them to think; secondly, to give them the fundamentals of our individual fields; and thirdly, to open their eyes to the importance of our subject in general and to the opportunities that it has for them. If a student can get these points in thirty-two hours he is, of course, just as well off as the other man who gets them in one hundred.

I am very glad to hear that Dr. Holt endorses what I have said in regard to the importance of this work. It really means putting the noses of the students into this work just long enough so that they can realize that the field is there. That is all we wish to attempt. On the other hand, if we do not do this, then the social workers and other laymen are going to control this part of public-health work. These individuals are always taking much second hand knowledge, and when you take second hand knowledge on a subject you are constantly in hot water. That is why, in reply to Dr. Talbot's statement regarding the aid that he has received from social workers in his dispensary, I must say that such work cannot be considered as good as that carried out by individuals who have also had a medical training—nurses. Unless we insist on the help of such workers, we are going to do the ideal piece of work that was so clearly presented to you last year by Dr. Gittings. Now, however, I would also like to say for Dr. Talbot that the Massachusetts General Hospital is at present giving this point much more serious consideration than any other hospital in the country, and it is planning to give to its nurses an excellent training in social-service work with the idea, I believe, that these nurses are in the future to replace the older type of social worker. Notwithstanding the fact that Dr. Cabot has a different, very high ideal of social workers, I believe that the nurse properly trained will be the better worker. As I have personally told Dr. Cabot I believe that the only individuals that would really fit his idea of social workers would be nuns and priests. I am not trying to belittle his conception of what the social worker must be, but I feel that nuns and priests are the only individuals that can hope to meet his idea of a social worker.

The remarks by Drs. Chapin and Kerley have really already been answered by Drs. Knox and Hamill. In infant mortality work the physician is needed to plan it and to direct the nurses and social workers. Of course the physician needs the nurses and other agencies—as the lawmakers—to help him accomplish this work, and that is why they are just as essential a part of an organization attacking the infant mortality problem as the physician himself is.

Dr. Morse pointed out that cooperation is essential. That is surely true. And I realize in presenting this scheme that it is impossible at present to expect to carry it out in Boston, New York, Philadelphia, and other cities where, owing to the existence of institutions for a hundred years or more, precedents have been established that are insurmountable. I realize that we have a much simpler problem in Cleveland and that we, therefore, can more readily and more easily get the cooperation and unity that are so desirable.

THE PATHOGENESIS AND PROPHYLAXIS OF RICKETS

JOSEPH E. WINTERS, M.D.
NEW YORK

Rickets is disordered nutrition. The most obvious and obtrusive change is retarded, erratic ossification. Suspension of assimilation of salts is the cause. Normal bone has 63 parts inorganic, 37 parts organic material. Rachitic bone 21 parts inorganic, 79 parts organic material. Its specific gravity is one-seventh to one-eighth that of normal bone.

At junction of epiphysis and diaphysis of normal bone, cartilage cells are incompletely straight tiers of six, eight or ten. Between each column is a definite line of salts. Rows of cartilage cells and an intervening line of salts, are mathematically regular. This layer of growing bone is $\frac{1}{24}$ of an inch in thickness.

In rickets, superimposed cartilage cells are fifty or sixty in each column. The columns are distorted and irregular. There is a mere sprinkling of salts, *in isolated nuclei.* This layer is $\frac{1}{4}$ to $\frac{1}{2}$ an inch in thickness.

Exuberance of cartilage cells, and *sparsely scattered islets of salts,* is the anatomical sign of rickets. The sole primary *pathological condition of rickets, is non-assimilation of salts in food.*

Whatsoever inhibits assimilation of salts in food causes rickets. That which insures assimilation of salts in food prevents and cures rickets.

Salts in food to be assimilated must be in organic synthesis with protein. In every food produced in the laboratory of nature, salts are in *organic* synthesis with protein. This is the anti-rachitic element in food. Disintegrate the synthesis of the great laboratory of nature and every food is a rachitic food.

Extremest rickets is with condensed milk as the only food for a prolonged period. For evaporation and condensation, milk is subjected to prolonged high temperature. Nature's synthesis of salts with protein is disintegrated.

Precipitate salts from milk, add these again to all the other constituents of milk, feed this to young animals for whom milk is a

sufficient, and a perfect food; they eventually die of inanition. *The unequivocal, anatomical sign of rickets is of every bone.*

Condensed, evaporated, desiccated milk, whey powder, every powdered dry preparation of milk is a rachitic food.

Rickets may be in any degree from just perceptible beading of ribs, to horrible deformity of entire bony framework. In milk, synthesis of salts with protein is light and easily impaired. Heat, according to degree and duration, *impairs,* or *disintegrates,* this synthesis. Synthetic impairment, as in pasteurization, and synthetic disintegration, as in condensation, cause different degrees of the same pathological condition. Pasteurized milk is a rachitic food.

Incipient, but positive rickets is with cereal in *young* infants. Diastatic ferments are in abeyance, during that which, by physiological edict, is the milk period. During period of total absence of amylolytic ferments, and of their gradual development between second and sixth months, assimilation of salts is inhibited by barley, granum, or any cereal. Salt curtailment causes rickets. At this early age rickets is commonly denoted by anemia, catarrh of mucous membranes, laryngismus stridulus, carpo-pedal contractions or general convulsions.

A child fed on condensed milk may be grossly fat and profoundly rachitic; with cereal, extremely emaciated, and markedly rachitic. With dry preparations of milk, malnutrition and rickets may be equally notable and obtrusive. With cereal as diluent of milk, impairment of nutrition may be inconsiderable; rickets just decipherable. Wheat flour, potash and extract of malt, cooked together, under the alluring name of "malt-soup," has been productive of a prodigious amount of unrecognized, small rickets. Cereal, dextrose, glucose, maltose, malt-soup, inhibit assimilation of salts in young infants and endanger rickets.

RICKETS AND COW'S MILK, WITH UNIMPAIRED ANTIRACHITIC ELEMENT

The unfounded assumption that large masses and bean-like masses in infant's stools were not casein, but fat, was most pernicious. Talbot, Southworth, Schloss, and others adduced conclusive scientific proof that these curds contain casein. The science of feeding was turned backward by dissemination of this groundless assertion. The sequel was, skimmed-milk, random, haphazard, hit-or-miss modification of milk. Consequent on this impossible food there ensued indiscriminate recourse to patented foods.

Of 200 feeding cases that came to the Vanderbilt Clinic Infants' Station, *the feeding previously prescribed by a physician,* 162 ordered one of the patented foods![1] The stultifying patented foods were a derision to all advanced pediatrists until they were the forced alternative for that portion of milk which is used in making skimmed-milk cheese—so tough and leathery that it is eaten only by Italian laborers!

These curds are formed in the stomach. When milk enters the infant's stomach, casein is precipitated and forms a solid clot. Digestion of protein is by gradual erosion. Therefore, the young infant cannot digest protein, unless it is in finely divided flocks, that there may be innumerable points for catalytic action of enzymes.

Precipitating casein enmeshes the fat! The coating of casein precludes action of lipolytic enzymes! Efficient action of proteolytic enzymes, and of lipolytic enzymes, is thwarted by these curds.

Neglect of physical behavior of protein superinduces rickets. Skimmed milk, when diluted with water and lime water as for feeding, and acidulated at feeding temperature, yields a few large, tough curds, which on stirring, stick together and sink.

The upper 7, 8 or 10 ounces from quart bottle of milk sixteen hours after milking, treated in same manner, gave a coarsely flocculent, floating curd, which can be broken up by stirring, but only with some difficulty, and *quickly collects into large masses again.* Protein masses cause intestinal irritation and diarrhea. Protein is drained off before time for assimilation. Protein contains the salts. Neglect of physical behavior of protein precludes salt assimilation. *Paucity of salts causes rickets.*

RICKETS AND CREAM

Centrifugal cream is the only available market cream. The constituent products of centrifuged milk are, fat-free protein, and protein-free fat. Centrifugal cream is protein-free, therefore, salt-free. Suspension of salts causes rickets. Salt starvation of centrifugal cream was erroneously diagnosticated fat indigestion, fat poisoning!

Skimmed milk, whole milk, upper 7, 8 or 10 ounces from quart bottle of milk, separately, or in combination with centrifugal cream, are rachitic foods.

1. Med. Record, Aug. 9, 1913, p. 241.

RICKETS AND FAT

Salts of calcium and magnesium are difficult of absorption. Free alkalies of the intestinal secretions split up neutral fats into glycerin and fatty acids. Fatty acids saponify salts of calcium and magnesium and thus render them readily absorbable.

Ample fat is requisite for salt assimilation for prevention and cure of rickets.

DIGESTION OF PROTEIN; DIGESTION OF FAT MADE FACILE

Top ½ ounce from quart bottle of milk sixteen hours after milking, diluted with water and lime-water as for feeding, and acidulated at feeding temperature, yields an immense number of finely divided, light, feathery flocks, which float in the solution, and are easily reduced to a semi-emulsified condition by stirring. Erosive action of proteolytic enzymes is perfect—ideal. Fat is not immured; action of lipolytic enzymes untrammeled; salt retention complete!

Erosive catalytic action of proteolytic enzymes; emulsification of fats by lipolytic enzymes; retention of salts, is dependent on and determined by, physical behavior of protein.

The prophylaxis of rickets, the key to all the enigmas of feeding, is to be sought in physical behavior of protein.

Disregard of physical behavior of protein is as preposterous as giving solid food before dentition.

RICKETS IN BREAST-FED CHILDREN

Proportion of salts in human milk diminishes progressively with increasing age of mother; they diminish progressively after the sixth or seventh month of lactation; there is diminution with frequent pregnancies, and when pregnancy occurs during lactation, there is early marked decrease. When mother's milk, owing to prolonged lactation, advanced age, constitutional feebleness, frequent pregnancies, or pregnancy during lactation, becomes poor in salts, *salt famine may cause rickets.*

SYPHILIS, TUBERCULOSIS, MALHYGIENE, AS ALLEGED CAUSES OF RICKETS

Syphilis.—A syphilitic infant, nursed exclusively by a vigorous young mother, never becomes rachitic. On rachitic food, a syphilitic infant seldom survives a sufficient period for rickets to develop.

Tuberculosis.—A child born of tuberculous parents, nursed exclusively by a healthy young wet-nurse, does not become rachitic, even though tuberculosis develop. On rachitic food, an infant with tuberculosis will almost surely succumb before time for rickets to develop.

Malhygiene.—Under most pestiferous hygiene a child nursed exclusively by a healthy young mother never becomes rachitic. Severe, acute rickets, in a squalid den, may be cured by supplying abundance of good breast-milk, without modification of hygiene.

Syphilis, tuberculosis, malhygiene, predispose to, but do not cause rickets.

The sole essential factor, *that which alone can cause rickets,* is the food factor. Rickets never develops except with rachitic food. Taint-free, hygiene faultless, food rachitic, a child invariably becomes rachitic.

The master's child in the mansion, family history unquestioned, hygiene utopian, with haphazard feeding, dies of extreme rickets, or, it ends in rachitic dwarfism, with unsightly, irremediable deformities, and is shackled and handicapped for life.

His servant's child, hygiene execrable, with an abundance of good breast-milk, is a joyous specimen of child life; makes a robust man, of enviable physique.

Studies in zoological gardens have demonstrated that rickets in young animals in captivity is always due to food which inhibits assimilation of salts. It is always cured by food which insures assimilation of ample salts, without change of environment. The sole etiologic factor of rickets in infants and young animals is wrong food. Malhygiene never causes rickets when feeding is correct.

25 West Thirty-Seventh Street.

DISCUSSION

DR. FREEMAN: It seems to me it is about time that we stopped considering rickets as a disease caused by food. While I believe that food may modify rickets to some extent, I do not believe that food is ever a cause of rickets. The worst case of rickets I ever saw was in a baby fed on good breast-milk; and so fed for about a year. The child was fat and well nourished but had a skull that could be dented anywhere, had wretched ossification, and died of pneumonia. It was the third child in the family and all three had had the same experience. The cause of rickets I believe, so far as the external cause is concerned, is asphyxiation. Our bad cases of rickets all occur in people that come from tropical climates. They get to a climate where they are shut up during the winter and rickets develops only during the winter. Rickets does not develop during warm months. In collecting a very considerable

number of cases from our dispensaries and institutions I have found that we got perhaps not quite as much rickets on breast milk, but I believe that we got more rickets in the babies fed on breast milk with accessory feedings, in one institution, than we did in any other class. My own feeling is that rickets is entirely a disease of bad air. It may be, perhaps, somewhat modified by feeding, but only secondarily.

DR. HOLT: During the last year and a half in the chemical department of the Babies' Hospital, we have been examining quite a large series of breast milks, with a view to the determination of the salt content. It takes a large amount of breast milk, at least a quart, to estimate the quantity of salts. Specimens may be obtained by combining the milk of many nurses to get an average result, or, by obtaining a sufficient quantity from a single nurse for such an estimation. With considerable trouble we have succeeded in getting twenty samples, from different women, large enough for an accurate salt determination. These analyses show that there is not that calcium reduction in advanced lactation that has been so often assumed.

DR. ABT: I would like to ask the essayist a question or two. The first is, How does Dr. Winters reconcile the prevailing view of phosphorus and calcium metabolism with the views that he adduces? The second is, What rôle would Dr. Winters ascribe to the internal secretions in the production of rickets? The third is, How would Dr. Winters explain the cases of undoubted congenital rickets on the basis of his theory of feeding? and another question is, How does he explain (or perhaps he has answered this) the cases in healthy babies, or babies that are fed by healthy mothers on breast milk?

DR. LA FÉTRA: There is another class of cases which those who have seen very many young babies must have been struck with, and that is the class of the very young and feeble babies, particularly premature babies, that tend to develop rickets notwithstanding the very best hygiene and the best form of breast-feeding we can give them. It is so commonly the experience that a baby that is born much under weight will develop rickets in spite of the best hygiene, that it seems to me there must be some deficiency in the infant that makes it tend to develop rickets. It has struck me, especially in the last three or four years, that no matter how well you may care for many of these premature babies, when they get a little older they develop rickets, and it does not seem to me that the food factor or even the factor of sunlight and fresh air enters very strongly into that particular type of cases. It looks to me as though it was something that did not come to the baby before birth, something in the internal secretions, perhaps, that must be a factor.

DR. GRIFFITH: Dr. La Fétra's remarks bring up a point which has long been in my mind; namely, the influence of inheritance in producing this disease. All the various other explanations which have been advanced do not seem to explain satisfactorily the special tendency of the negroes and of the Italians to develop rickets. I cannot see that they are any more poorly fed or any more exposed to bad air than are children of other races in this country. This matter of inheritance was studied some years ago by some European physicians who looked into the history of families who had children with rickets, and found that there were repeated instances of its having been present in the parents. I recall one family under my observation in which three breast-fed children, under my own care and under the best hygienic surroundings, developed rickets; and on inquiry I found that the mother and her brother had been knockkneed when children, to an extent which

caused their playmates to make fun of them. I have seen other children developing rickets, also fed on an abundant breast milk of perfect character so far as indicated by chemical examination. I am convinced that, although food probably plays a part in the production of the disease, it is no more the sole cause than is the influence of bad air. That inheritance is an etiologic factor is, as I said, certainly to be considered, although I do not believe it is yet proved. I agree with others that no definite cause has yet been discovered.

DR. TALBOT: I have studied a hundred cases of rickets with the help of intelligent social workers from all these points of view, and after having put all my figures together, could find no cause for rickets.

DR. WINTERS: Congenital rickets I have never seen. A great deal has been written against the possibility of congenital rickets. I can conceive, however, that it might occur. The blood condition of a mother during pregnancy might be such as to produce rickets in her offspring. In fact, considerable has been written regarding the diet of the mother during pregnancy and its influence on the development of rickets in the embryo.

As to diminution of protein and salts in breast milk during lactation and all the other conditions alluded to in my paper, there is probably nothing more thoroughly established in medical science than these facts. Chemists in every part of the world have been uniform in their statements as to diminution of protein and salts with advancing lactation.

As to the influence of phosphorus in rickets, that question was answered in the paper. Rickets in colored children and in Italian children one sees constantly at college and dispensary clinics, and season has no influence on its occurrence; if there is a seasoned influence, there is more rickets in summer than in winter. Change of environment and climate is not a factor in the development of rickets in these classes. As to its occurrence in these classes, when breast fed, a single afternoon in a dispensary class will convince one that these mothers *invariably* give their nursing babies all kinds of pernicious foods. For thirty-odd years I have been connected with the Demilt, and the statements of nursing mothers are so at variance with what one sees in the dispensary examining room that one becomes convinced that it would require a large colony of social workers to get at the truth as to what the Italian and colored nursing mothers give their infants in the way of vicious food.

THE EFFECT OF THYMUS EXTIRPATION

JOHN HOWLAND, M.D., ROY D. McCLURE, M.D.,
AND
E. A. PARK, M.D.
BALTIMORE

The first extirpation of the thymus gland was undertaken by Restelli about seventy years ago. He tried to remove the thymus gland from a large number of animals, but failed to obtain experimental results, because his animals did not survive the operation. His work is therefore of only histological interest.

The great impetus to the study of the function of the thymus was given by the fundamental work of Friedeleben. Friedeleben investigated the thymus function from the experimental, clinical and pathological standpoints. He performed numerous experiments on animals, chiefly dogs, some of which survived his operations and developed changes which he attributed to the absence of the thymus gland. His work first suggested a relationship between the thymus function and the growth of the skeleton, an idea which later von Bash and Klose developed.

Since Friedeleben's time much experimentation has been done to determine the nature of the thymus function, notably by von Bash, Fischl, Klose and Vogt, Matti and Nordmann. Dogs, rabbits, guinea-pigs, sheep, goats, chickens and frogs have been used as experimental subjects, but the most important results have been obtained in dogs, for the other animals are less suited, and some of them totally unsuited, for complete thymus extirpation. The results of all experiments, including those in dogs, have been widely different in the hands of different observers, and furnish an experimental literature as confused and difficult of interpretation as that surrounding the question of splenic function.

Von Bash concluded that the thymus was not essential to life, but that its removal produced changes of a transitory nature in the skeleton and in the growth and activity of the animal. Klose and Vogt, on the other hand, came to the conclusion that the thymus was absolutely essential to life. They found that, following extirpation, their dogs

passed through definite stages of adiposity, cachexia and coma, to death, developing extreme rachitic changes in the skeleton, degenerative changes in the brain and muscles, hyperplasias of the thyroid and adrenal, and an abnormal blood picture. Klose and Vogt's views were modified, but in general sustained by the work of Matti. Fischl, Nordmann and numerous other investigators, on the other hand, have obtained essentially negative results from thymus extirpation.

Our work is not yet complete, so that the present report is of a preliminary character. We have failed to find any absolutely constant changes in our dogs following thymus extirpation. In the majority of the animals autopsied there have been found changes in the thyroid gland resembling those of the human thyroid in Graves' disease. The adrenals of the operative animals were, in most instances, found to weigh more than the adrenals in the controls, but without the disproportion in size between medulla and cortex described by Matti. The bones of our animals failed to show any changes suggestive in the slightest degree of rachitis, nor did the operative animals seem less active than their controls. Three of the operative animals lagged considerably behind the controls in weight, an observation which may be of significance, and, on the other hand, may be due merely to the effect of a severe operation, or to chance. We are certain, however, that the thymus gland is non-essential to life, and that no such picture follows thymus extirpation as that portrayed at so much length and skill by Klose. The majority of our animals were operated on before the twenty-first day of life, and have been under observation for a year.

DISCUSSION

DR. FRIEDLANDER: In explanation of the difference of opinion with reference to the extirpation of the thymus, it should be borne in mind that some of the German observers have reached conclusions somewhat similar to those just obtained by our Baltimore investigators; and one explanation which has been given, which has doubtless occurred to these gentlemen too, is that it is very difficult to extirpate the thymus completely, for the reason that it has been found that in many animals there are accessory thymi which very often hypertrophy after the large thymus is extracted, and may take the place of the original thymus and thus prevent the effects of the thymectomy. With reference to the experimental work which has just been reported, I feel that it is only fair that we should call attention here to the fact that thymectomy as performed in the human being is an exceedingly dangerous operation and that there is, so far as we can now see, no absolute occasion for doing thymectomy in cases of enlarged thymus.

The last complete report of the surgical procedure was that by Parker, reported last year; in fifty-odd cases there was 33 per cent. fatality. Using

the method of shrinking the thymus with the Roentgen ray, we have now had twenty-seven cases with one death; and it is only fair, I think, that we should bear in mind that this method is exceedingly simple, and that it is safe, and that there is now no justification for thymectomies in the human being.

In corroboration of this, it should be borne in mind that Veau, who with Olivier worked out the technic of thymectomy (and Veau, by the way, did more thymectomies than any other single operator), announced in the Paris Pediatric Society that he had given up thymectomies; that he would shrink the large thymus with the Roentgen ray; and I believe that we are justified in claiming that by means of the Roentgen ray we have a means at hand for the control of enlarged thymus—incidentally for cases of status lymphaticus—which is just as efficacious, in my opinion more efficacious, and certainly safer than thymectomy.

DR. ABT: What does Dr. Friedlander consider a large thymus? One that produces symptoms or one that shows itself to be large by physical examination?

DR. FRIEDLANDER: Dr. Lang, who with me has been working on Roentgen-ray diagnosis of the enlarged thymus, is perfectly convinced, as I am, that the diagnosis of the large thymus by the ray alone is insecure. I, for one, am not willing to take the roentgenogram alone. I consider that we have a right to say we have an enlarged thymus if we can map out the enlarged thymus by percussion and that is not at all difficult with many cases when we have the associated symptoms of stridor, the cough and the dyspnea, if in conjunction with this you have a roentgenogram which is confirmatory.

I should like to call to the attention of the Society that we have been able to show that very frequently there is a large, flat thymus, which does not give a shadow with the Roentgen ray; and it is only in cases of what we call the "cap" thymus that the shadow is apt to be distinct. We are perfectly sure that the clinical evidence is of more value than the roentgenogram, which latter I should regard as merely confirmatory.

DR. PARK: Serial sections were made of all tissues resembling thymus from the neck and thorax of the experimental animals, and rests found in only one instance. Klose emphasizes the importance of thymus rests in or about the thyroid gland, and hypothecates the presence of thymus rests in these situations to explain negative experimental results. As a matter of fact, thymus rests at these points must be very infrequent, at least in the dog, as we have failed to find any in a single instance.

I have no knowledge in regard to the dangers of the partial removal of the thymus gland in human beings, but cannot believe that the operation need be a dangerous one. Certainly, it is hardly fair to cite the statistics of thymectomy in the published series of cases of so-called thymic asthma as a proof of the dangerousness of the operation, because those patients are usually extremely ill when operated on and die from other causes than the operation. I should suppose, from post-mortem examinations of the thymus, that in the normal individual the partial removal of the thymus gland would be an exceedingly simple operation, and I have been told that such was the case by surgeons. In the more recent operative treatment of certain types of Grave's disease it has been the custom at the Johns Hopkins Hospital to remove a part of the thymus coincidently with the resection of the thyroid without increasing to any extent the risk to the patient.

THE DIAGNOSIS AND TREATMENT OF "LATE" HEREDITARY SYPHILIS *

BORDEN S. VEEDER, M.D., AND P. C. JEANS, M.D.
ST. LOUIS

RELATION OF "EARLY" TO "LATE" SYPHILIS

During the past two years we have seen 123 cases of manifest hereditary syphilis at the St. Louis Children's Hospital. Of these seventy-four, or 60 per cent., belonged to the group commonly described as "late" hereditary syphilis while only forty-nine, or 40 per cent., were infants under 1 year of age with the classical symptoms of rash, coryza, and enlarged spleen which characterize the "early" type. The division into "early" and "late" is arbitrary and only a matter of convenience, as these "early" symptoms may first develop after infancy, while "late" lesions may develop early in infancy. The "late" lesions may also develop primarily in older children without any "early" lesion having occurred or been recognized. Thus lesions comparable with the secondary (eruptive), tertiary (gummatous), and quaternary or para-syphilitic vascular and nervous lesions), stages of acquired syphilis, are included among the clinical manifestations of "late" hereditary lues.

In only twelve of our cases were we able to obtain a positive history of an "early" lesion in infancy, while in twenty-five cases the history was definitely negative in regard to this point. In the remainder the history was indefinite or could not be obtained. It is highly probable that some of our forty-nine "early" cases will later develop other symptoms and thus ultimately belong to the group under consideration. In connection with the large group of cases without early lesions it is interesting to note a study[1] of the Wassermann reaction in 101 infants in St. Louis (thirty-three of whom were from this clinic) who showed no clinical evidences of syphilis. But two positive and one doubtful reactions were obtained. Although this series is small, it would tend to show that the latent infections in infancy are not so extremely common in St. Louis as might be imagined from our figures.

1. Blackfan, Nicholson and White: AM. JOUR. DIS. CHILD., 1913, vi, 162.

We are now making a study of latent infection in older children by testing the blood and examining the brothers and sisters of the patients in our series. A large percentage of such children have given positive Wassermann reactions although they have never shown clinical evidences of luetic infection. Many items of interest have occurred in these familial studies which will be reported later.

GENERAL STATISTICS

The ages of the seventy-four patients were as follows:

Years of age.....	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12
No. of cases.....	6	4	6	4	6	4	10	7	5	5	11
Years of age.....	12-13	13-14	14								
No. of cases.....	2	2	2								

Eighty-three per cent. of the patients were white and 17 per cent. colored, while of the total clinic attendance 92 per cent. are white and 8 per cent. colored. Thirty-three were males and forty-one were females.

We were able to obtain a definite history of the feeding in infancy in twenty-seven cases. Twenty-three had been breast-fed and only four artificially fed. This may be looked on as an illustration of the better chances for life of the breast-fed syphilitic infant over the artificially fed, as the ratio is much different from the ratio of the breast to bottle-fed infants in general.

CLINICAL LESIONS

The following list of clinical diagnoses shows the wide variety of lesions encountered. In many cases there were multiple lesions, and the following figures give the number of cases in which each lesion occurred. In every case there was a positive Wassermann reaction which was checked in practically every instance by a second test.

Bones—	
Periostitis tibia	3
Periostitis skull	1
Joints—	
Acute arthritis knee	7
Acute arthritis ankle	1
Skin—	
Macular eruption	1
Alopecia	2
Condyloma anus	3
Gummata	3
Central nervous system †—	
Mental deficiency	14
Cerebrospinal syphilis	8
Hemiplegia	5
Epilepsy	4
Paralysis	3
Chorea	2
Hydrocephalus	2
Ulcerations—	
Nasal	2
Laryngeal	1
Pharyngeal	1

Eye—		Ozena	1
Interstitial keratitis	17*	Enlarged spleen (only lesion)	1
Choroiditis	1	Torticollis	1
		Aortitis	1
		Obscure abdominal pain	1
		Obscure pain in legs	2

* 23 per cent.

† Thirty-two patients in all or 43 per cent.

The large number (43 per cent.) of our patients with lesions of the central nervous symptoms is one of the most interesting features from the standpoint of diagnosis. During the past two years we have tested the blood of thirteen patients with hemiplegia admitted to the wards of the hospital of which five reacted positively. In one case—a girl of 5 years—admitted at the onset of the trouble, we were able to limit the extent of the lesion by a vigorous treatment. Some of these cases had been previously looked on as poliomyelitis. Two of the eight cases reacting negatively were unquestionably birth injuries. Of eight cases of chorea tested (following and including the first positive case whose history is given later), two gave positive Wassermann reactions. In three of the negative cases there was a definite history of rheumatism, while no history or evidence of rheumatism was noted in the two positive cases and in the three remaining negatively-reacting cases.

Mental deficiency was usually associated with some other condition as epilepsy or cerebrospinal syphilis. During the period covered by our cases there were thirty-three cases of mental deficiency admitted to the wards of the hospital of whom fourteen were syphilitic. This is a very much higher percentage than is usually found and does not express the true relation of mental deficiency due to syphilis to mental deficiency in general, but is the result of admitting cases for this particular study.

Among the more unusual lesions were an aortitis and a torticollis. Three patients with indefinite pain of obscure origin—once in the abdomen and twice in the legs—which had persisted irregularly for some time and for which no cause could be found, gave positive Wassermann reactions and the pain disappeared under specific treatment. The cases of acute arthritis were very interesting. Some patients were sent to us for tuberculosis and others for acute articular rheumatism. One case (knee) developed in a child with a tuberculous hip who had been under observation and treatment in the orthopedic department for several years.

The absence of Hutchinson's teeth which is so generally described as a common symptom of syphilis was very noticeable in our cases. In forty-eight patients who were old enough to have permanent incisors the teeth were notched in but three instances. Ill-shapen and deformed teeth were very common. They were usually small and peg-shaped with broken, irregular surfaces, and were separated widely from one another and without good alignment. We have also observed typical Hutchinson's teeth in a non-syphilitic patient.

It will be noted that most of our cases fall into one of three large groups. The first of these is made up of eye conditions (24 per cent.) and is so well recognized that it needs no discussion. The second comprises lesions of the central nervous system (43 per cent.). So many of these cases have given positive Wassermann reactions when there has been nothing to suggest lues in the early or family history that we now make the test as a part of the routine examination of nervous cases. The third group is in many ways the most interesting as it contains a group of cases less frequently recognized as of a possible syphilitic origin. It is made up of cases of chorea, acute arthritis, torticollis, and obscure muscular pain—in other words clinical conditions which are often grouped together as "rheumatic"—and amounts to about 20 per cent. of our cases. It has long been recognized that these acute "rheumatic" conditions are the result of bacterial toxins, and our cases would indicate that syphilitic infection of an hereditary nature is one of the common causes of these cases developing in childhood. We have checked the diagnosis of these cases by repeated blood tests and by Wassermann reactions on the parents. The strongest proof that they are the result of syphilis and not an accidental association lies in the rapidity with which the lesions improve under antispecific treatment.

TREATMENT

The treatment we have gradually evolved consists of a combination of neosalvarsan and mercury. We prefer neosalvarsan to salvarsan because of the simplicity of the technic, which is an essential factor in dealing with children. The neosalvarsan is dissolved in 1 c.c. of freshly distilled water for each decigram and injected intravenously into any available vein with an ordinary glass hypodermic syringe. The entire preparation of the drug and syringe and the administration can be accomplished in from twelve to fifteen minutes. We made

a number of intramuscular injections earlier but soon gave them up because of the pain and discomfort to the patient and the objections of the parents. The dose of the neosalvarsan varies according to the age and clinical condition of the patient—as a whole we give larger doses than are usually advised and have had no unpleasant or bad results. At first the injections were followed in a few instances by nausea and vomiting which we are inclined to attribute to the use of a "sterile" water instead of a freshly distilled water, as we have not observed such sequalae in the last year and a half. Although it is claimed by competent observers that neosalvarsan is less effective than salvarsan, we prefer neosalvarsan because it is effective and fulfills certain essential requirements for use with children which salvarsan does not. For the mercurial part of the treatment we have come to use gray powder exclusively in the late cases and to a very large extent in the early cases. For the treatment of acute lesions (eruptions, ulcers, arthritides, etc.) the neosalvarsan is far more effective than inunctions of mercury, while the gray powder gives very good results and can be given over longer periods of time with less disturbance than mercury in any other form. We have had no experience with the mercurial injections which are so effective in the acquired forms of syphilis, as intramuscular injections are not practical with children, at least in the class of patients that we are treating.

The course of treatment we use varies somewhat, but with the usual case presenting an acute lesion is in general as follows: Three or four intravenous injections of neosalvarsan are given with a gradually increasing dosage two or three days apart. Then mercury is started, with small doses, which are gradually increased until the patient is taking a fairly large dose. In a few weeks this is interrupted for a short time and then repeated. In a number of cases a second and a third series of neosalvarsan injections are given alternating with the mercury. The mercurial treatment is continued for an indefinite period of time according to the requirements of the individual case and in general until a negative Wassermann reaction is obtained, if this is possible.

Forty of the seventy-four cases have been treated in this way and observed for a period of from three months to two years. As the cases differ so much in the types of lesions and length of treatment, they cannot be summarized to any advantage and the results

of treatment are best seen by abstracting a few typical cases illustrating different points. We have given over two hundred intravenous injections of neosalvarsan and have never observed any bad or unpleasant effects besides those mentioned above.

EFFECT OF TREATMENT ON ACUTE LESIONS

There can be no question as to the value of neosalvarsan in acute syphilitic lesions with the exception of interstitial keratitis. In the acute joint lesions, ulcerations, general convulsions, torticollis, etc., the effects seem immediately to follow the first intravenous injection.

L. B., No. 3725, a girl of 17 months, was admitted for backward development. She had been normal until two months previously when she had an attack of convulsions. At the time of admission she was unable to stand alone and was apparently blind, not even noticing the milk bottle or a light. Attacks of general convulsions were frequent. Wassermann reactions on the blood and spinal fluid were positive. Neosalvarsan was given 1-14-13, 0.5; 1-17-13, 0.3; 1-22-13, 0.2 intramuscularly. The child was much better by this time and was beginning to notice objects. No attacks of convulsions since first injection. 1-31-13, 0.2, intravenously; 2-4-13, Wassermann positive; 2-12-13, 0.15, intravenously; 2-20-13, 0.2, intravenously.

Beginning about the first of February, 1913, there was rapid improvement and by the middle of the month the child was playing almost as a normal infant. She was put on gray powder and on March 27 was discharged with a negative Wassermann. On April 24, one month later, there was a sudden return of symptoms and two days later she was readmitted. Between 12 o'clock noon and 9 p. m. there were ten attacks of general convulsions. She was then given 0.2 neosalvarsan intravenously which immediately stopped the convulsions. Inunctions of mercury were pushed for a month but no more neosalvarsan was used as the patient remained free from convulsive attacks. On May 17 the Wassermann reaction was negative and the patient was again discharged. In March, 1914 (fifteen months later), she was apparently normal although there have been "slight attacks of convulsions at times." She has taken gray powder intermittently.

R. C., No. 5043, a girl of 12 years, was admitted with a maniacal type of chorea about three weeks after the onset. She was unable to stand or walk and could not sit on a chair unless supported. Restraint was even necessary when she was in bed. Speech had been lost for several days. A family history of syphilis together with Hutchinson's teeth in the patient gave the idea of an underlying luetic infection. A dose of 0.4 gm. neosalvarsan was given a few hours after admission and a marked effect was noted within forty-eight hours. It was repeated two days later when a Wassermann reaction was strongly positive. Inside of a week the patient was up and about the ward and had lost most of the choreic symptoms. Neosalvarsan and mercury were continued for some time.

L. D., No. 4072, a 14-year-old girl, was admitted for acute pain and swelling of the right knee. One week later the ankle became swollen. A Wassermann taken two days previous to the involvement of the ankle was strongly

positive. Neosalvarsan, 0.5, was given and in two days the swelling and pain had disappeared.

N. D., No. 3715, was admitted for an interstitial keratitis of five weeks' duration and an acute arthritis of the knee of two weeks. Wassermann, three plus positive. On Dec. 29, 1912, an injection of 0.9 neosalvarsan was followed by rapid improvement of the knee, but in a few days it became swollen and painful again. On Jan. 1, 1913, 0.9 neosalvarsan was given and followed by rapid relief. Treatment was continued with no return of the arthritis, but the interstitial keratitis showed but little improvement.

E. G., No. 6489, 8 years, admitted March 2, 1914, for acute arthritis of the right knee. Wassermann on March 5 was four plus positive. Neosalvarsan was given on March 7 and by March 11 all pain was gone from the knee and motion was good. She was given more neosalvarsan and discharged. Six weeks later the patient returned with an acutely swollen and painful left knee and the Wassermann was still four plus positive.

T. B., No. 3791, a boy of 6 years, was admitted with an extensive laryngeal ulcer of three weeks' duration. He was given five injections of neosalvarsan and within two weeks the ulcer had healed.

From these few reports the rapid action of neosalvarsan may be seen when the lesion is acute. The exception is apparently an acutely developing interstitial keratitis. In this condition it has an influence in some cases, but in our experience a long and extensive course of mercury is necessary in order to obtain much improvement.

TREATMENT AND CHRONIC LESIONS

We have been able to secure but little permanent effect from neosalvarsan nor have we observed any advantage from its use in chronic cases of the late type; that is, where a lesion such as a cerebrospinal syphilis or an interstitial keratitis has been present for some time. As a matter of fact the most intensive treatment with combined neosalvarsan and mercury has been of little avail in these cases.

H. W., No. 3492, a boy of 14, was admitted for cerebrospinal syphilis (fixed unequal pupils, staggering gait, dementia, etc.), which was said to have first shown itself three years previously. Wasserman four plus positive. He was given 0.5 neosalvarsan in October, 1912, which was followed by inunctions of mercury. An injection of 0.9 neosalvarsan was given in December and the inunctions continued. In February, 1913, he was given two injections of neosalvarsan of 0.5 each and then gray powder was continued for several months. It was without effect on either the clinical condition or the Wassermann reaction. From time to time he has taken gray powder in large doses, but his condition to-day is worse than when treatment was started over twenty months ago.

In this same group must be placed the cases of hemiplegia following vascular changes and other conditions where structural changes have occurred. The only possible result of treatment in such cases is the prevention of further lesions.

TREATMENT AND THE WASSERMANN REACTION

It is not within the scope of this paper to enter into a theoretical discussion of the Wassermann reaction. For clinical purposes we look on the Wassermann reaction as a symptom of syphilis, comparable to an arthritis, a keratitis, a periostitis, etc. It is the most common symptom and present in such a large percentage of all clinically positive cases that we are inclined to be skeptical about the diagnosis of late syphilis in any untreated patient who has a negative Wassermann reaction. For example, we have had a case of interstitial keratitis in a boy who had marked Hutchinson's teeth. The Wassermann was repeatedly negative but because of the two classical symptoms of hereditary lues he was given a thorough antisyphilitic treatment which, however, was without effect. Later a rapid and marked improvement of the eye condition followed the use of tuberculin and in our opinion there is no reason for looking on this case as one of syphilis. A positive Wassermann reaction may be the only symptom of syphilis for many months or years (latent hereditary lues) and then other symptoms develop, as is shown both by the early history of some of our cases and the tests we have made on brothers and sisters of patients with manifest lues. It is quite difficult to get these patients with latent cases to take a course of treatment, but there is no reason why this symptom should be treated any less vigorously than a manifest lesion. Nothing definite is known of the future of these latent cases with a positive Wassermann and we are attempting to follow some which have not been treated.

The Wassermann reaction is stronger in hereditary lues than in any other form of syphilis and hence is correspondingly more difficult to make negative. Not only is this so but it is very apt to become positive again if treatment is stopped when it has been made negative as the result of treatment. We have not determined quantitative effects of the treatment on the Wassermann reaction.

N. D., No. 3715, a girl of 8 years, was admitted in December, 1912, for interstitial keratitis and acute arthritis. Wassermann four plus positive. The arthritis improved rapidly following two injections of neosalvarsan of 0.9 each followed by one of 0.5 and another 0.4. Gray powder, gr. 1 three times a day,

was then given for three months when inunctions were started and continued for several weeks as the eyes did not improve. In May, 1913, the Wassermann was still four plus positive. Gray powder has been taken almost continuously since at the limit of tolerance and at times potassium iodid has been given. On Jan. 16, 1914, the Wassermann was still four plus positive and again on May 19, although the child has been given an intensive and uninterrupted antisyphilitic treatment for eighteen months and the eyes have improved considerably.

C. M., No. 53166, aged 13 years, was first seen on July 16, 1913, for an interstitial keratitis which began one month previously. Wassermann four plus positive. He was given 0.5 neosalvarsan intravenously every other day for three doses and then put on gray powder. Three months later the Wassermann was still positive and the gray powder was continued. On March 6, 1914, the Wassermann was still four plus positive, although the eyes had improved very much with eight months' continuous treatment.

K. V., No. 4081, a boy of 7 years, with an acute keratitis, had a positive Wassermann on Sept. 23, 1912, at which time he could not distinguish fingers at 5 feet. He was given neosalvarsan 0.3 and inside of a week was able to make out pictures across a room. Two more doses of neosalvarsan were given, but as on March 14, 1913, the Wassermann was still strongly positive, three more injections of 0.4 each were given. A Wassermann taken five weeks later, or May 2, 1913, was still four plus positive. Gray powder — 1 grain three times a day — was then given for three months but was without effect on the Wassermann. Eyes almost entirely well. This Wassermann, Aug. 1, 1913, was ten months after beginning treatment. The gray powder was then pushed to a point of salivation and kept up almost continuously until a *negative* Wassermann was obtained on Jan. 5, 1914, after fifteen months of vigorous treatment. Treatment was discontinued for two months and then a course of mercury given for one month. On May 19, 1914, six weeks after this was finished, the Wassermann reaction had again become *positive*.

At times it is not difficult to obtain a negative Wassermann reaction as in the following case.

G. G., No. 3443, an infant of 18 months, who was admitted for ulcers of the face and fingers (gummata) had a positive Wassermann Oct. 15, 1912. There was extremely rapid improvement following an injection of 0.22 neosalvarsan into the buttocks. This was repeated in fifteen days and the infant discharged one month later with a small nodule in the cheek the size of a pea. No other treatment was given. Five months later a Wassermann was taken which was strongly positive and the patient was given three intravenous injections of neosalvarsan of 0.25 each and discharged as there were no clinical lesions. Eight months later — fourteen months after first admission — the Wassermann was negative and in April of this year there had been no return of symptoms. This was one of the few cases with a permanent result following neosalvarsan alone and is decidedly exceptional in our experience.

In a treated case or a case under treatment in which the Wassermann has become negative, other symptoms of syphilis may develop, showing that the disease cannot be regarded as cured or quiescent from a negative Wassermann reaction alone.

E. P., No. 3995, a boy of 4½ years, was admitted for paronychia and malnutrition. No history obtainable, but the Wassermann was three plus positive on March 7, 1913. He was given seven intravenous injections of neosalvarsan (2 gm. in all) between this date and April 23, 1913. Inunctions of mercury were given to the point of salivation. There was marked improvement following the first few injections of neosalvarsan. In June and again in September he was given a course of mercury and on Oct. 24, 1913, (seven and one-half months after the first Wassermann) the Wassermann reaction was negative. In March of the present year he developed an alopecia which has become quite extensive. A Wassermann reaction on April 11, however, was negative, and again a few weeks later after a provocative dose of neosalvarsan.

SUMMARY

We have found the incidence of manifest hereditary lues of the "late" type much greater in proportion to the incidence of "early" syphilis than previous figures would indicate. The largest group of our cases (43 per cent.) exhibited lesions of the central nervous system, but we particularly wish to call attention to a group of cases with lesions associated as "rheumatic"—chorea, acute arthritis, torticollis, myalgia. By Wassermann reactions and the "therapeutic test" we have found that such lesions are not infrequently due to an hereditary syphilitic infection.

It has been our experience that acutely developing lesions respond promptly to intravenous injections of neosalvarsan, but that in order to obtain any permanent results and prevent the recurrence of symptoms an intensive and long continued mercurial treatment must be given in addition. The syphilitic infection—as measured by the Wassermann reaction—is most persistent, and although an intensive and uninterrupted antisyphilitic treatment has been given for nearly two years we have not been able to obtain a negative Wassermann reaction in some of our cases. Moreover, the Wassermann reaction has usually returned when treatment has been interrupted in those cases in which it has become negative as the result of treatment. We do not feel that it has been demonstrated as yet that an hereditary syphilitic infection of the "late" type can be eradicated.

St. Louis Children's Hospital.

DISCUSSION

DR. KERLEY: I am very glad to hear Dr. Veeder's paper as to the results of late hereditary cases. I have had experience with only three in private practice, in which we have been unable to get a negative Wassermann by using the neosalvarsan repeatedly intravenously and in association with mercury. I thought it was my fault or some peculiarity of the patients. The others showed the usual bone involvement.

DR. HAMILL: At the last meeting of the Association of American Physicians, Dr. Warthin read a paper on the persistence of the spirochetes in the tissues of syphilitics who had had no clinical evidences of the disease, and in whom a negative Wassermann had continued for several years. The subjects had died of other diseases and the tissues were examined post mortem. It was chiefly in the heart, aorta and testicles that he found the spirochetes. These findings he considered suggestive confirmation of the view that syphilis is probably an incurable condition. Dr. Welch asked how he explained the cases of syphilis which were apparently cured and later reinfected through the medium of the primary lesion as in the original attack. This opens up an interesting field for discussion; but the point that I wished to make is that this evidence of the persistence of the spirochetes bears a suggestive relationship to the results of Dr. Veeder's work since he has reached the conviction that none of the type of cases that he has been discussing are definitely cured.

A CASE OF ACUTE CHYLOUS ASCITES (NON-FATTY, PSEUDOCHYLOUS, LACTESCENT OR MILKY TYPE) IN A BOY EIGHT YEARS OLD

FRANCIS HUBER, M.D.
NEW YORK

OPERATIONS AND SURGICAL COMMENTS BY
HENRY M. SILVER, M.D.
NEW YORK

In a recent most valuable, thoroughly scientific and exhaustive monograph Dr. S. Gandin of St. Petersburg, discusses the different types of chylous effusions in serous cavities. The work is based on the study of three medical and two surgical cases, and a critical review of 281 references covering the literature up to date.

The classification chylous, chyliform and milky, non-fatty forms of ascites, is not universally adopted. Many of the more recent writers make no distinction between the chylous and chyliform varieties; others prefer to retain the designation chyliform (which does not necessarily refer to the origin of the fluid), some again do not make any distinction between the fatty and the non-fatty ascites, regarding them, in contradistinction to the chylous, as chyliform or pseudochylous.

This confusion has led to a further distinction of mixed types, chylous-adipose, chylous-pseudochylous and adipose pseudochylous effusions.

1. The *true chylous* is due to the presence of chyle. 2. The *chyliform or fatty,* though resembling the first, owes its milky-like appearance to emulsified fat, the result of a fatty degeneration of cellular elements. 3. Finally, in the third variety, the opalescence is not due to the presence of fatty emulsion, but is caused by some opalescent substance, the exact nature of which is still undiscovered.

Chylous ascites is due to the escape of chyle from the duct or receptaculum chyli, the result of trauma or disease processes, malignant or benign. The specific gravity varies from 1.007 to 1.033, average 1.0158. The reaction is alkaline or neutral.

The fluid resists putrefaction, is without odor, does not clot, but may separate into layers on standing, the fat appearing on top. This

variety is distinguished, first, by the finely divided state of the fat, and secondly, by the absence of leukocytes or other cells showing fatty changes.

The chylous and chyliform effusions have been carefully studied, not only as to their composition, but also from the pathological standpoint. Wallis and Scholberg (1910) believe that there are no characteristic pathological anatomical changes.

Kelly (1907) would restrict the name to cases in which injuries or ulcerations of the chylous system have been found.

Witlin (1909) believes that chylous ascites or chylothorax can only be explained by trauma.

Between these extremes there are those who would explain the occurrence by stasis or evidence of pressure on the thoracic duct; others find macroscopic changes somewhere in the chylous system; finally some base their diagnosis on microscopic changes in the vessels, or on the grounds of an individual anatomical predisposition.

Undoubted cases of a rupture (trauma or result of disease processes) in some portion of the chylous system, have been reported as of occasional occurrence.

Mechanical obstructions in any part of the course of the duct from the receptaculum chyli to the left subclavian vein, or in some of the tributaries in the mesentery, the result of cicatricial contractions, new growths, cysts or tumors, may give rise to stasis and transudation through the weakened and dilated walls. In some of these cases diffuse dilatations of the main duct or entire chylous system were found; in others more or less localized distention of the vessels in the mesentery and intestinal walls existed, depending on the situation of this obstruction. It must be remembered that pathological conditions which lead to a gradual interference in the ductus are more readily compensated through the collateral circulation than those of acute origin, a circumstance which may explain the rarity of cases.

Chyliform or fatty (adipose) ascites differs from the above. It is not due to leakage of chyle but to the formation of fat in the effusion; the globules are larger and cells containing fat are present, specific gravity 1.009 to 1.026; average, 1.01625. It is usually associated with intra-abdominal new growths or with chronic peritonitis, simple or tuberculous.

The origin of the fatty, lactescent type, has been attributed to fatty degeneration of cellular elements, a lipemia, etc.

The non-fatty, pseudochylous, lactescent or milky type is differentiated, as the name implies, by the absence of fat. Specific gravity 1.005 to 1.030; average, 1.0132.

1. Such an effusion does not lose its milky appearance after being shaken up with ether, benzol, chloroform, etc., even though rendered alkaline beforehand.

2. When fat is present the amount is too small to give rise to a milky appearance.

3. The microchemical reactions of fat (specific color, effects of heat and fat solvents) are negative.

4. Precipitation of the albumen leaves a clear solution.

5. Finally in a number of cases the opalescence is presumed to be due to minute particles of albumen, nucleo albumen, mucoid, lecithin, lecithoglobulin, etc. Gandin,[1] who has carefully reviewed the voluminous literature and weighed the evidence, does not believe that the negative results of the microchemical reaction of the so-called pseudochylous fluid invalidate the assumption of the fatty nature of suspended molecular granules.

In considering critically and at length the various properties (physical, microscopic, chemical, and physiological) of chylous, chyliform and pseudochylous fluids, Gandin[1] is unable to establish any important differential characteristics; furthermore, he does not think that we are justified in the use of the above-mentioned classification. A thorough critical review of the pathogenesis of the chylous and pseudochylous types leads to a similar result. His conclusions based on his studies and experiments, are briefly as follows:

GANDIN'S CONCLUSIONS

1. The addition of even small percentages (0.01 to 0.1 per cent.) of the finely emulsified fat of chyme or homogeneous milk to a transparent fluid gives rise to a milky cloudiness.

2. The milky appearance of an effusion in a serous sac is due to the presence of emulsified fat.

3. It has not been demonstrated in a satisfactory manner, that the presence of any substance excepting emulsified fat, can give rise to the milky appearance. The presence of emulsified fat in milky

1. Gandin, S.: Patogenese und Klassification der Milchartigen Ergüsse, Ergebn. d. inn. Med. u. Kinderh., 1913, xii, 219.

effusions, without the admixture of chyle is not probable, nor has such been demonstrated.

4. As chyle represents the only recognized source of finely emulsified fat in the body, milky and opalescent effusions must be regarded as chylous.

5. The terms "chyliform" and "pseudochylous" are not applicable as distinct pathogenetic entities, consequently they are superfluous.

6. Effusions, containing fat in drops, but not in an emulsified form, do not present the typical milky appearance.

The term fatty (adipose), introduced by Quincke, is applicable to such cases.

CASE REPORT

History.—S. H., male, aged 8, was admitted to Beth Israel Hospital Dec. 15, 1913. He was born in New York and had never been in any of the southern or eastern countries.

The family history has no bearing on the case. He had measles when 7 months old, and what was supposed to have been a nephritis, with edema of the extremities, clearing up after four weeks illness, at age of 4 years. With the exception of pneumonia at 5½ years, the boy was reported to have enjoyed fair health up to the time of his present illness.

Careful questioning failed to elicit any history of trauma. Two weeks before admission the child felt generally ill, suffering from a severe nasal discharge, though he continued to attend school. A week later he vomited undigested food, since which time his appetite has been poor. Two days before he entered the hospital the father on dressing the child noticed an increase in the size of the abdomen and a swelling of the genitals. No fever, cough, headache, general weakness or urinary disturbances were present.

Physical Findings.—The patient was fairly well developed, somewhat anemic and rather poorly nourished. No evidences of prostration, dyspnea, no petechia or other eruption or glandular enlargement were noticed.

Heart and lungs negative. Abdomen symmetrically enlarged, fluctuation wave readily transmitted from side to side. Careful palpation failed to reveal a tumor or any swelling in the abdominal cavity; no tenderness or rigidity present. The veins were markedly distended over the lower half of the abdomen and right upper quadrant; also over the upper parts of both thighs. The center of the abdomen was tympanitic; the flatness in the flanks, changing quickly with a change in the position of the patient, showed a large amount of free fluid in the abdominal cavity.

The edema of the penis and scrotum was quite marked. There was slight edema of the thighs, but none about the feet or ankles; abdominal walls and back slightly infiltrated. The temperature was 100 F. It quickly fell to normal and remained so. The pulse, which was 120, rapidly came down to 80, varying between 80 and 100; at times 110. The respirations were 24 to 30. Red blood-cells, 4,700,000; hemoglobin, 70 per cent.; white blood-cells, 9,800; lymphocytes, 16 per cent.; polynuclears, 79 per cent.; eosinophils, 4 per cent.; mastcells, 1 per cent; urine negative. Von Pirquet negative on repeated occasions. Wasserman also negative.

Two weeks later red blood-cells, 3,500,000; hemoglobin, 65 per cent.; white blood-cells, 10,000; lymphocytes, 11 per cent; polynuclears, 88 per cent.; eosinophils, 1 per cent. Phenolsulphonephthalein test for renal efficiency 30 per cent. on one occasion and 20 per cent. the second.

Though the general condition remained excellent, appetite "simply enormous" with regular bowels, the abdomen became larger, the diaphragm being pushed up as high as the middle of the scapula on the right side, and lower angle on the left. Edema of the genitals and thighs, particularly the left, also increased; the subcutaneous tissues of back and abdominal walls pitted on pressure. Notwithstanding distention there was relative little discomfort or dyspnea. The urinary excretion was greatly diminished, about 8 or 9 ounces daily, though allowed at least 20 ounces of fluids. Urine negative, no casts, cells or bacteria.

Management.—Jan. 6, 1914, the abdomen was tapped in customary manner; over 3,000 c.c. of a slightly turbid, milky fluid were removed to relieve the distention. A considerable amount, approximately half, was allowed to remain. Microscopically a few lymphocytes and some finely granular matter with brownian movements were found. Evaporating a drop on the slide, crystals of sodium chlorid were detected. A bacteriological examination failed to reveal any organisms. No sugar or fat present.

EXAMINATION OF ASCITIC FLUID

From S. H.

Quantity about 500 c.c.; color and appearance, milky, still turbid after filtration; specific gravity, 1.011 (20 C.); reaction, neutral; total solids, 1.25 per cent.; material in suspension, 0.016 per cent.; ash, 0.72 per cent.; organic matter, 0.53 per cent.; sodium chlorid, 0.64 per cent.; sulphates, *nil;* urea, *nil;* sugar, *nil;* albumin, 0.319 per cent.; primary proteose, 0.055 per cent; secondary, also present, unestimated; globulins (indications ?) not estimated; fats, absent. LOUIS E. SALAS, M.A., B.Sc.

For four or five days subsequently, considerable oozing took place through the small puncture, necessitating a change of dressing several times daily. The edema of the scrotum and abdominal walls diminished. As soon as the slight wound had healed, the abdomen refilled rapidly and the edema of the back, abdomen and thighs also increased, necessitating a second tapping. January 17 over 2,000 c.c. were taken away. It presented the same opalescent appearance, similar to that removed before. The rapid recurrence of the ascites, it seemed to us, demanded more radical measures. If the abdominal fluid, rich in proteins, salts, and the characteristic constituents of the tissues and body fluids could be diverted to the blood and lymph streams, the patient certainly would gain in every way. After a consultation with my surgical colleague, Dr. H. M. Silver, an exploratory laparotomy was decided on, first, with a view to a clearing up the nature of the lesion, and secondly, to determine the possibility of draining the "peritoneal pond" into the subcutaneous tissues of the thighs and abdominal walls. This part of the subject will be taken up by Dr. Silver to whom my sincere thanks are tendered for his interest and skilled surgical aid in the care of this interesting case.

Diagnosis.—A careful consideration of the physical condition and the history of our patient did not enable us to hazard any suspicion as to the etiology. In the absence of any traumatism, the rapid appearance of ascites, the large prominent veins with local edema of abdomen and genitals, pointed to some

intra-abdominal pressure, though no tumor or other swelling could be detected, even after tapping. The general appearance and bearing of the child were against malignancy.

The character of the fluid obtained by tapping, its milky opalescent hue, and the absence of cells filled with fat, justifies a diagnosis of chylous ascites (third variety), non-traumatic in nature and of uncertain origin.

DISCUSSION

A majority of the writers believe that the chylous origin of the fluid may be established or recognized clinically, first, by the "test of Strauss"; secondly, by the rapid return of the fluid after tapping.

The Strauss test is based on the fact that the amount of fat in the chyle depends on the diet. Thus, if a larger amount of fat is allowed or a smaller percentage is taken in, corresponding changes will be noticed in the fat contents of the fluid removed in repeated tappings at regular intervals. Though confirmed by some, the results obtained by other observers have been rather conflicting.

The rapid accumulation of the fluid noted in our case and in others reported in literature, has been the subject of comment by many authorities.

It was observed by Vermage in 1700, and confirmed by many writers subsequently.

Senator attributes the rapid recurrence to stasis, either in the portal system or in the chyle-duct or lymphatic circulation. Others conclude that there has been a rupture of some portion of the duct or one of the tributaries, permitting the escape of chyle into one or other of the serous cavities. On the other hand, instances have been recorded in which the fluid did not return quickly. And furthermore we should not forget that in other pathological states, as advanced cases of cirrhosis of the liver, in which the question of chylous ascites does not enter, a rapid return of the fluid is not unusual.

Aside from the presence of the chylous fluid, the case is interesting in many other respects. For instance:

1. The small amount of urine passed. Though only 8 or 9 ounces were excreted in twenty-four hours, there was no evidence of any systemic disturbances, as headache, nausea, vomiting, etc.

2. The general well being of the patient. Excepting the discomfort due to the distended abdomen, the boy did not make any complaint. The appetite was good and bowels regular. Pulse and temperature normal.

3. The rather unusual type of ascites with edema of the back, abdomen, thighs and genitals (ankles, legs and chest, not involved) and no edema of the face at any time throughout the course of the disease.

4. The rapid return of the fluid after tapping, presumably explained by the fact that a collateral circulation had not been established, as condition was rather acute.

5. The marked diminution in the amount of edema of abdomen, genitals, etc., after the tapping, and its rapid return when the puncture had healed.

6. The rapid subsidence of the external localized edema after operation.

7. The decided increase in the urinary excretion soon after operation, evidently the direct results of the removal of the intra-abdominal pressure and the establishment of peritoneal drainage.

8. The brilliant success of the surgical measure adopted.

OPERATION WITH SURGICAL NOTES

HENRY MANN SILVER, M.D.

Early in January of this year (1914) Dr. F. Huber requested me to see and operate on a boy 8 years old, suffering from chylous ascites. As a careful history of the case has been given above, it will not be necessary to repeat it. Although the patient was in rather poor condition, and the fact was recognized that ascites patients have an impaired vital resistance, deficient powers of repair, and do not stand well extensive operations under general anesthesia, it was decided to do an exploratory laparotomy as quickly as possible under ether anesthesia.

Exploratory Laparotomy.—An incision beginning just below the umbilicus and extending downwards for 3 inches just to the right of the median line was made through the layers of the abdominal wall. When the peritoneum was opened considerable fluid escaped and about 1,500 c.c. were drawn off by the aid of the suction apparatus.

The coils of small intestines presenting in the wound were greatly distended with gas and very pale, and on careful examinattion of all the coils within reach a most interesting condition was observed. The lacteal vessels, not only on the intestines, but in the mesentery, were much enlarged, being flexuous in their course, constricted at intervals and presenting a beaded or varicose appearance. The constrictions on the intestines were so tight in places that the lacteal vessels would disappear only to reappear in the mesentery. Enlarged glands were found in the mesentery, small near the intestines, but growing larger as they passed to the root of the mesentery where they became very numerous.

Some were about the size of a small hickory nut, soft, elastic and of a yellowish color. It was not deemed advisable to remove one for the purpose of closer study and microscopic examination, as the child's condition was not good. As careful an examination was made of the organs within the abdominal cavity as was possible through a 3-inch incision. Nothing abnormal was detected. Before closing the wound, the question of what form of drainage should be resorted to, presented itself.

The ascites was chylous, came on quietly, the child had not been living with any tuberculous patient at home, gave no history of injury, and there were no evidences of compression of the portal vein, as the intestines and omentum were pale and showed no sign of congestion. As it seemed to me that the ascites was due to compression and distention of the lymphatic or chylous vessels by the large number of enlarged glands within the mesentery, some simple form of internal drainage which could be quickly done was called for. This would relieve the intra-abdominal pressure until the child's general condition could be improved by good food, fresh air, sunlight and possibly some form of medication. Moreover, it would conserve a fluid rich in proteins, salts and the characteristic constituents of the body fluids; the little boy had already lost over 5,000 c.c. of this fluid withdrawn at two previous tappings.

The internal drainage was carried out as follows: Six strands of No. 7 white silk 4 inches long were caught in the grasp of a narrow-bladed dressing forceps, carried into the abdomen and thrust through the peritoneum to the outer side of the femoral vessels into subcutaneous tissues of the thigh, only half an inch of the silk remaining within the peritoneal cavity. This was repeated on the opposite side. At the upper angle of the wound six more strands of silk of the same length as used below were thrust into the subcutaneous tissues of the abdominal wall above the umbilicus, the lower half inch remaining within the abdominal cavity. Great care was taken to allow only a very little of the silk to project within the abdominal cavity, as it has been found by actual experiment that long silk strands may cause intestinal obstruction by forming attachments to the omentum or any organ within reach.

Course.—The patient reacted well and his convalescence was rapid and uneventful. The dressings were removed at the end of a week and the wound was found completely healed, with no edema of its edges. There was some edema in the upper part of the right thigh, very litttle in the left thigh, and none over the upper portion of the abdomen. The patient was allowed to be up and about the ward early in order to maintain the circulation of the body fluids by exercise, especially in the portal and lymphatic systems. The boy was discharged from the hospital in fine condition the latter part of March. No evidences of fluid within the abdomen could be found; there was no edema of the thighs or abdominal wall; no thickening of or about the silk drains could be felt.

Five months after operation the boy was found to be in excellent health; the fluid had not returned. Abdomen lax, no masses could be felt on a most careful examination, nor could any thickening be made out in the neighborhood of the silk drains.

Although Lambotte[2] of Antwerp, was the first to use silk to drain the abdomen, it was not until Mr. W. Sampson Handley[3] published his

2. Lambotte, E.: Semaine méd., 1905, xxv, 19.
3. Handley, W. S.: Brit. Med. Jour., April 16, 1910, p. 925.

work on the use of silk for artificial lymphatics in cases of brawny arm in the last stages of breast cancer, also its use in draining fluid from the abdominal cavity, that surgeons began to give this method of drainage any attention. Handley from an experience gained from an interesting case of ascites which showed but little edema of thighs and upper abdominal wall after operation, draws the following inferences:

(1) Without causing edema, drainage into the thigh has gone on since the operation; (2) if the absorptive powers of the tissues are normal, and the amount of fluid led into them is not excessive, the appearance of edema is not to be expected.

McDill[4] in order to determine the exact changes that took place in the silk used for abdominal drainage carried on a number of experiments in animals. He found that the short intra-abdominal ends showed a densely organized membrane of connective tissue outside of the silk and a general infiltration of the meshes by cells, single, in bundles, and in septa; although the angle of the silk with the peritoneum is plainly marked, showing an actual ectropium of the peritoneum along side of the silk which really acts as a silk connective tissue plug. This angle is the place at which McDill claims that an intraperitoneal fluid must find its exit by pressure and gravity to the subcutaneous lymph spaces, along the outside of and not within the body of the silk.

4. McDill, J. R.: Surg., Gynec. and Obst., Nov., 1913, p. 523.

MEGACOLON AND MICROCOLON *

LANGLEY PORTER, M.D., AND ALANSON WEEKS, M.D.
SAN FRANCISCO

Pediatricians express difference of opinion as to the relative frequency of megacolon. Some authors of wide experience are able to make reports on only one or two such cases; that such experience is not general is attested by the fact that in the literature of the past four years there are more than one hundred papers each of which deals with one to six cases. It follows, therefore, that the affliction cannot be extremely rare. We have encountered six cases during the past five years; two of these passed from observation after one view and, therefore, are not included in this report. Microcolon, on the other hand, seems to be truly an unusual condition, as our search through the literature revealed but three certain and one doubtful case, although it seems probable that some reports under the caption "Stenosis of the Colon" may have referred to this condition.

The name "megacolon" has been used to describe the clinical picture characterized by obstinate constipation, great abdominal enlargement, visible intestinal peristalsis with the constitutional results of toxemia due to fecal retention. Constipation at times causes such retention of feces that fecal masses accumulate; these may shift and cause attacks of acute or subacute obstruction of the bowel. The literature referring to this congenital enlargement of the colon is ample. In his masterly article written in 1908, Finney presents a bibliography of 206 titles compiled by A. W. Fisher, complete to the time of his writing. We have searched the libraries diligently and find that over 100 papers on the subject have appeared since Finney's writing. One of the best of these later articles was written by Barrington-Ward and published in January, 1914. One must agree with Barrington-Ward's statement that Finney's paper so completely discusses all phases of the disease that it must remain the authoritative communication on this subject for a long time to come.

Although Finney shows that some twenty odd cases, one by Lewitt of Chicago (1867) and another by Jacobi, were clearly described

* From the Medical Department of Leland Stanford, Jr., University.

before Hirschsprung published his studies of the malady in 1896, we owe the eminent Dane a debt for centering attention on the importance of the condition. This writer classified giant colon as: (*a*) true megacolon, first noticeable at birth or early infancy; (*b*) pseudomegacolon, making its first appearance in adult life and showing dilatation of the colon with hypertrophy of its muscle, and arising secondary to some partial obstruction that came on during the postnatal period. Many writers deny pseudomegacolon pathologic validity, and, in fact, there seems to be little common to the two conditions.

Barrington-Ward divides the theories which seek to explain the origin of megacolon into four classes: (1) the mechanical; (2) the neuropathic; (3) the inflammatory and (4) the congenital. Under the mechanical theory he arranges five subheads: (*a*) the view of Barth, that an increased mesentery permits torsions of the sigmoid with partial occlusion and subsequent hypertrophy and gigantism of the intestine; (*b*) the view of Marfan, that the increased length of the intestine, especially the sigmoid portion, throws the bowel into a loop which causes kinking obstructions; (*c*) Treves' view, that the obstruction follows atresia of the anus and rectum; (*d*) Perth's idea, that valve formation is the cause of the obstruction; (*e*) Wilkie's conception, that the primary obstruction is due to the distention of the pelvic colon with meconium at time of birth or shortly after. As will be seen, none of these theories makes the difficulty of interpretation less.

Four different explanations are offered in elucidation of the neuropathic theory. It is suggested: (*a*) that there is a neuromuscular defect of the segment of the intestine; (*b*) that there may be a paralysis of a segment of the intestine; (*c*) that there is some implication of the sympathetic nervous system, and (*d*) that reflex spasm of the sphincter ani, a result of a fissure in ano, is the basic cause of the trouble.

Adherents to the third theory hold that a colitis that later becomes chronic originates those pathological processes which afterward produce the full picture of megacolon.

The proponents of the fourth theory assign the only cause which seems to explain the known facts. They hold with Hirschsprung that the condition is an anomaly of development and that the hypertrophy and dilatation both occur in the fetal intestine before birth. Concetti, while acknowledging the congenital origin of the dilatation, considers that the hypertrophy follows effort made to overcome the obstruction

due to an inert segment of the dilated bowel. Other writers, among them Fenwick, believe that the hypertrophy is the congenital feature, and that the dilatation is the result of the pressure of the retained gases. In favor of the theory of congenital origin are age at onset, presence of constipation and other symptoms from birth as illustrated in all the cases here reported as well as by a study of the pathology. Furthermore, cases have been reported in which fully developed megacolon occurred in prematurely born children.

Barrington-Ward points out that developmental errors are most common at the junction of the proctodeum and hindgut and that the site of next greatest frequency is at the junction of the foregut and midgut. He considers that in megacolon the developmental error occurs at the former site, and he draws attention to pyloric stenosis as a possible example of developmental error in the later position. It is well known that the increase in the length of the intestine during the last month of intra-uterine life is usually about 25 per cent., but that there is great individual variation in this respect. At birth the infant's intestine is at least one-tenth longer in proportion to the body length than is the adult's. Much of this increase goes to make the sigmoid of excessive length in the infant. In the normal baby, growth rapidly balances this relationship, but in cases of giant colon such balancing is impossible, at least in well-developed cases. From such facts Finney infers that the colon is still under morphogenetic evolution, that the inconstancy of its length and minor details are but variations in the progressive evolution toward the final arrangement, and that the anomalies are but reversion types. According to him, in megacolon all the segments of the intestine may be involved, but more often the condition concerns that segment whose evolution is most primitive. He lays great stress on the thickness of the mesocolon in one of his cases. This he states to have been the result of enlarged lymph-glands together with enormously dilated lymph and blood vessels, which corresponded exactly with the gigantic section of the bowel. This appearance suggests to him a state of hypernutrition and he calls attention to the fact that this same increase in vessels is a feature of that gigantism which occurs in the limbs; he suggests that the conditions in the bowel may be parallel. A reference to the photomicrographs will show that in our cases very large much-dilated vessels occurred not only in megacolon, but in pyloric stenosis and in giant small intestine as well. But none of our material showed the lymph-node hyperplasia that Finney found in his case.

Briefly stated, Barrington-Ward's theory, which seems the most explanatory, is that there is a congenital abnormality in the lower end of the hindgut which takes the form of a muscular hyperplasia. It usually involves the rectum and spreads a varying distance up the pelvic and iliac colon. It may be that the whole of the hindgut is affected, but in some cases there is a definite localization of the change and the hyperplasia affects only a segment of the intestine. The hyperplasia of the muscles gives rise both to thickening of the walls and to increase of the bowel lumen. The physiologic functions of the intestine are in abeyance, and blocking is produced in the bowel behind, for a varying distance, which depends on the extent and duration of the dilatation and hypertrophy. In time this fecal stasis produces necrotic changes in the mucosa, an ulcerative colitis follows which is the cause of death in most instances. It seems reasonable to us that Barrington-Ward and Finney each contribute facts that, taken together, explain the underlying causes of the pathology in these conditions.

CASES OF MEGACOLON

The following cases of congenital enlargement of the intestine are reported because they serve, some to throw light on the disputed points, and others to illustrate the danger of temporizing management, while the last case shows the efficacy of operative interference. The case of microcolon is included not only because of its rarity, but because it illustrates a condition opposite to megacolon and at the same time furnishes us with an abnormality in the small intestine which in every way parallels the pathological changes found in the large intestine in cases of giant colon. We present for comparison the photomicrographs of the giant colon, of the hypertrophied small intestine and of a case of hypertrophic pyloric stenosis. A comparative study of this picture will show how nearly identical the muscle and blood-vessel changes are in the three conditions. Our cases of megacolon are as follows:

CASE 1.—Baby S., referred by Dr. A. B. Spalding. Seen on the fourth day of life. The mother and father were healthy people. The birth normal, at full term. The child was brought in because of increasing abdominal distention and because the bowels had not moved since just after birth, when a small amount of mucus and meconium-like material was passed. No one had noticed the passage of any flatus.

On admission, the child weighed 8 pounds. It appeared somewhat dried-out, the skin hanging loose and lusterless. The child was well formed with no deformities except that the abdomen was exceedingly prominent. This

enlargement was especially marked when contrasted with the dried-out limbs, and was taken to be due to gas retention. It was noted that there was an area of visible intestinal peristalsis which passed across the abdomen from the left iliac fossa to the border of the ribs on the right side. This area was horse-shoe-shaped and seemed to be due to movements in two separate segments of the intestine. The upper segment showed movement from left to right, while the movements in the lower segment appeared to pass from right to left. On examination the heart and lungs were normal; no tumors were determined in the abdomen. The abdomen was distended but not hard and was easily pal-

Figs. 1 and 2.—Patient 3, three months before death from pertussis.

pated. The anus was patent; the little finger entered an apparently normal rectum but was arrested just above the brim of the pelvis by what felt like a narrow band.

The child retained milk from one-half to one hour and then rejected it all. Attempts to give enemas were unsuccessful; the water returned uncolored. A bismuth meal was given and the child radiographed by Dr. Boardman. In the picture the stomach appeared normal, but the plates taken one and two

hours after the meal showed no retention of bismuth in either stomach or intestine; all had been vomited. The child was kept alive by hyperdermoclyses of 4.5 per cent. glucose in normal salt solution.

Based on the finding in the rectum of a definite narrowing and of an apparent obstruction to the exit of the bismuth meal from the stomach together with visible peristalsis, a diagnosis was made of multiple stenosis of the intestinal tract with narrowing at the duodenum in the small intestine and at the pelvic colon.

A surgeon was asked to see the child and to attempt multiple anastomosis. When the abdomen was opened the true state of affairs was revealed. A giant colon bulged into the wound and was returnable with great difficulty. The

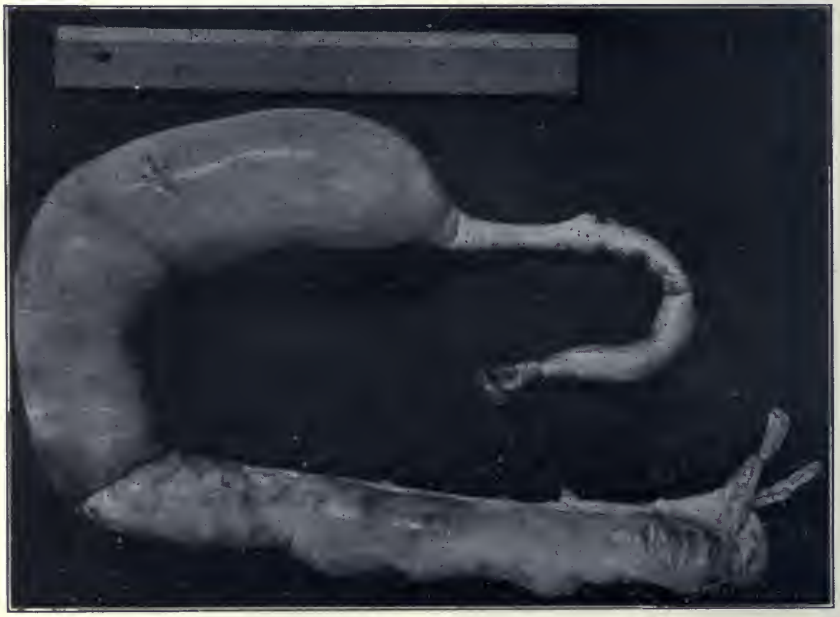


Fig. 3.—Colon from Case 3.

area of peristalsis proved to have been produced by a long redundant loop of the sigmoid which stretched across the abdomen. The outgoing loop crossed the transverse colon as high as the pylorus; it then wound around the lower border of the liver, and the incoming loop, starting above the hepatic flexure, ran thence to the left iliac fossa, where it terminated in a distinctly narrow but slightly hypertrophied area about 1½ inches in length. The child succumbed to the operation. Necropsy confirmed the findings revealed at the operation.

Unfortunately, the removed intestine was lost, hence it is impossible to report on the microscopic conditions present in this specimen.

This case is of interest in that it shows true giant colon to be a condition that may present itself at birth with fully developed dilatation and hypertrophy—this in spite of the view of those authors who hold the dilatation to be variable and secondary, and others who have a like opinion about the hypertrophy. It is further of interest because,

Fig. 4.—Intestines from case of microcolon. *A*, gigantism of small intestine; *B*, microcolon.

had the diagnosis been made and the intestine promptly opened for drainage, the child might have survived, and a later attempt could have been made to remove the large intestine, as has been done by Arbuthnot Lane. The way in which young children respond to gastro-

enterostomy for pyloric stenosis is warrant for the faith that this child might have survived operative interference.

CASE 2.—A. H., 18 months of age. Seen in the course of private practice with the history of having had obstinate constipation and "big stomach" since the fourth month. He was the first and only child, born at full term, after protracted labor. The child had been difficult to feed in that he had not thriven on varying formulas and was astoundingly constipated. On one occasion, during the sixth month, he had passed several days (the mother said ten), without evacuation, and on many occasions there were periods of from two to five days without bowel movements. At such times the abdomen became tremendously enlarged beyond its ordinary huge size and the child would suffer

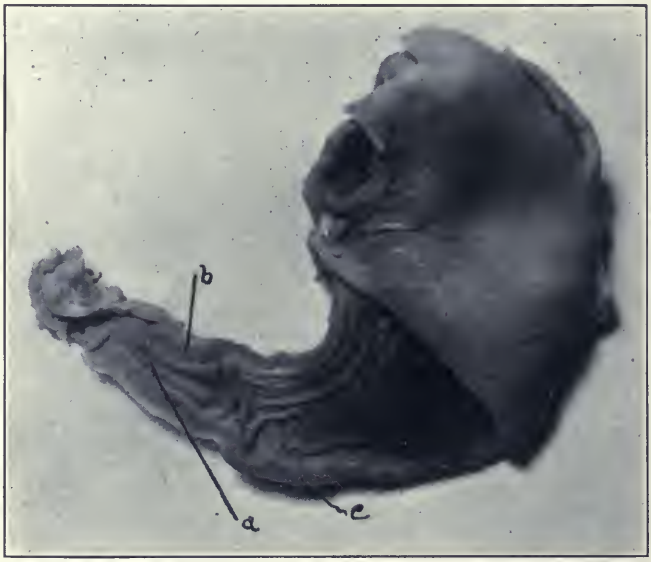


Fig. 5.—Hypertrophic pyloric stenosis, from photograph, full size; *a,* narrowing of lumen; *b,* ring muscle; *c,* hypertrophy of circular muscular coat of stomach. The narrow lumen of the pylorus is seen to be almost completely occluded at *a.* The wall of the pylorus is seen to be greatly thickened at *b,* and is continuous with the hypertrophied musculature of pyloric antrum.

attacks with rapid, shallow pulse, embarrassed breathing, and threatened collapse. The mother had learned that by the aid of a soft rubber catheter she could manipulate the contents of the bowel and so greatly relieve the child of the accumulated gas, and with this maneuver she carried the patient through many such crises. She had also discovered that the blocking of the bowel by feces was relieved and the gas most readily passed when the child's hips were held high above the shoulders. It is possible that in this position the retained mass was freed from the narrowed sigmoid by the aid of gravity.

On examination there was revealed an emaciated child with a huge abdomen, measuring twenty-four inches at the umbilicus, and looking, as one colleague remarked, like a balloon tied to a little skeleton. The skin was dry and hung in folds everywhere except from the abdomen. The distention of the abdomen was exaggerated at the flanks, where the bulging was extraordinary. The diaphragm was high, and the costal region very wide. No visible peristalsis was noted. Masses, hard but doughy, were felt in the abdomen, one the size of a large orange on the right above the navel, another in the left iliac region about the same size, and smaller ones lay in what seemed to be the line of the transverse colon. These were moved about and felt with difficulty because of the extreme distention. Rectal examination revealed a wide, lower rectum, the little finger was stopped at about the pelvic rim by what seemed a definite narrowing. Bimanual palpitation defined a tumor in the left iliac region, but pressure could not force it downward through the constriction.

Fig. 6.—High-power photomicrograph of the hypertrophied ileum. The normal mucosa is seen at *a*, under which lies the somewhat thickened submucosa. The muscular layers (*b* and *c*) are hypertrophied, especially the internal coat. The serosa, lying external to the muscular layers (*c*) is edematous.

The child now entered the Lane Hospital and it was decided to wait twenty-four hours and then to create an artificial anus. The patient seemed in excellent condition. Glycerin and water enemas were ordered and an attempt was made to produce evacuation. Several such enemas were given without result in the course of twelve hours and each time all of the fluid was returned unstained, according to the nurse's report. Apparently, however, a mass of feces in the transverse colon was dislodged and dropped down into the narrow part of the intestine, where it produced an acute obstruction. At about 10 p. m. on the day of the child's entrance into the hospital, one of us was summoned

hurriedly to find the child *in extremis* with the pulse running shallow and uncountable, the face gray, the lips blue, and the patient barely conscious. There was the most tremendous distention conceivable. In the absence of Dr. Weeks from San Francisco, a hurried summons was sent to another surgeon, but before he could reach the operating-room the child was dead. Obviously, the thing which should have been done was to incise, draw out the intestine and open. In the absence of surgical experience, and in the hope of finding skilled aid at once, one unskilled in surgery hesitated to assume this responsibility, but, undoubtedly, if such an emergency were faced again, the opening would be made without hesitation.

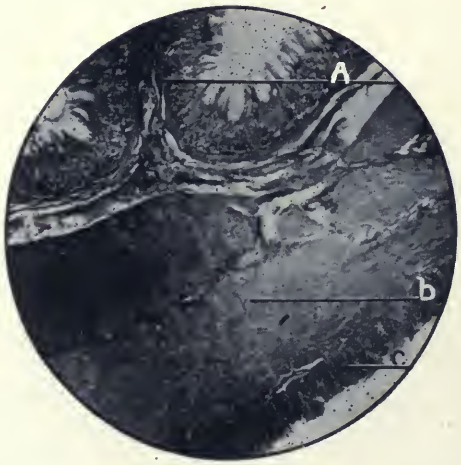


Fig. 7.—Photomicrograph of hypertrophied wall of pyloric antrum. The mucosa (*a*) is normal and is covered with a layer of mucus. Beneath it lies the submucosa, which is loosened from the underlying internal muscular coat (*b*), which shows a marked hypertrophy. Throughout it are seen fibrous connective tissue strands extending down from the submucosa. The external muscular layer (*c*) is not much thickened.

Necropsy was denied us in this instance, so our only help from it lies in the illustration of the potential dangers of the condition and in the emphasis it puts on the view that the only safe procedure in these cases is to make a preliminary colotomy with artificial anus at the earliest possible moment, so that the intestine may be emptied of its retained material. After such procedure the poisoning ceases, the child's nutrition improves, and, with a wait of from two to four months, the physical conditions are so mended that there is warrant for a resection of the entire large intestine with the hope of permanent cure.

CASE 3.—E. S., male, 19 months of age, weight 15 pounds and 4 ounces, seen in consultation with Dr. Dudley Smith (Figs. 1 and 2). Full term, normal birth, birth weight 7¾ pounds. The abdomen was so distended at birth that the parents thought the child's hips were deformed because of the great contrast. Constipation at that time was very marked, and there was no evacuation until the fourth day of life, when scybalous masses of hardened meconium were passed, but in small quantity. In the second month a period of nine days elapsed without any movement of the bowel; the abdomen was greatly distended, and the child was much distressed. Evacuation was finally accomplished by enemas and castor oil, but these agencies produced only small marble-like scybala. At the time of this attack no signs of collapse supervened with the distention. The physician then in charge considered the condition to be due to unsuitable food, and the child was subjected to numerous changes in diet

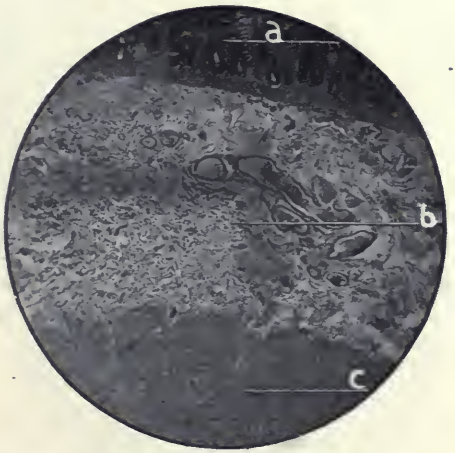


Fig. 8.—High power photomicrograph of megacolon. The mucosa at (*a*) is normal. The submucosa (*b*) is markedly thickened and fibrous and contains many large dilated vessels filled with blood. The muscle layer (*c*) is also thicker than normal.

without effect. He gained slowly in weight and strength, but such attacks as just described recurred almost every month. On one occasion in the fourth month there was marked diarrhea which lasted for a week and was accompanied by depression and great distress, which was relieved by castor oil and turpentine enemas. At six months, after obstruction of four days' duration, a characteristic attack supervened with pain, distention and collapse; at the eighth month another similar attack came on.

Dr. Dudley Smith now saw the child for the first time. He was able to effect evacuation of the bowels and to keep the child in fair condition by large doses of magnesia and the passage of a soft rubber catheter, which effectively relieved the distention. The child's nutrition began shortly to improve; his weight curve commenced to ascend and he reached 19¼ pounds in the seventeenth month, in spite of the fact that severe obstructive attacks occurred during the fourteenth and sixteenth months. During the eighteenth month the

boy's weight fell during such an attack to 15¾ pounds, when one of us saw him for the first time and we coincided with Dr. Smith's opinion that operative interference held the only promise of relief for the condition. This help was not to be, however, as the child was even then in the first stage of pertussis, to which he later succumbed. The necropsy revealed a typical megacolon (Fig 3).

Figure 9

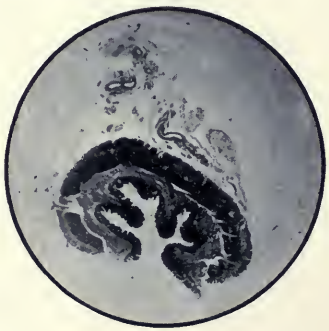


Figure 10

Figs. 9 and 10.—Low-power photomicrographs of the microcolon (Fig. 10) and the dilated and hypertrophied ileum (Fig. 9) showing the relative size of the gut of the different levels and the thickness of its walls. The marked dilatation of the ileum (Fig. 9) masks to a certain degree the hypertrophy of the muscular coats so that they do not appear much thicker than those of the microcolon (Fig. 10).

The physical examination of this child showed simply a distended abdomen in a rickety subject who exhibited visible peristalsis over the middle and upper section of the abdomen; there were palpable fecal masses, and the rectum on examination revealed a narrowing at the pelvic brim. This case, like the last, shows the method of obstruction that occurs in this pathologic state and in each instance, the pain, collapse, and obstruction were relieved after the passage of a hard, narrow fecal mass which apparently had lodged in the narrowed sigmoid.

CASE 4.—E. P., male, 22 months of age, referred by Dr. Hughes of Sonoma, Cal., with the diagnosis of Hirschsprung's disease. This was the second child, full term, normal birth. Parents and other children normal and in good health.

Fig. 11.—High-power photomicrograph of microcolon. The mucosa (*a*) is normal, and the underlying submucosa is somewhat fibrous. The muscular layers (*b* and *c*) are relatively thicker than normal, but the decrease in the size of the intestine is evident in all its layers, no single layer being decreased at the expense of the others.

Symptoms were not noticed by the parents until the fourth month of life. From that time the history was like that of the other cases reported, malnutrition and difficult digestion with attacks of obstruction and collapse. On examination we were confronted with a yellowish gray-skinned anemic child, weighing 22 pounds. There was an enormous abdomen, which measured thirty inches at the level of the umbilicus. We saw no visible peristalsis on this occasion, and there was no such tenseness of the abdomen as had been met in the other cases. Palpation of the abdomen revealed several tumors; the largest, the size of a fetal head, lay in the right iliac fossa, extending upward and toward the right where the bulging was marked. Other similar

masses lay above the umbilicus and to the left of it. The largest mass was smooth, easily movable and was taken to consist of feces lying in the caput coli. Rectal examination revealed no narrowing; on the contrary, the rectum and colon were very large and roomy. Previously, a diagnosis of tuberculous mesenteric adenitis had been made. Bimanual examination with the finger in the rectum revealed a smooth, velvety peritoneum and contradicted this assumption.

Dr. Weeks now saw the child for the first time and advised operation, proposing a two-stage procedure. At the first operation the abdomen was opened on the left side and an artificial anus was created. The method of Levison of San Francisco was utilized and the retained fecal masses were then evacuated and we found that the one which had been discovered on the right side was lying, not in the caput coli, but in the redundant loop of the huge sigmoid. This giant sigmoid crossed over the abdomen and filled most of it; it overlay the apparently normal caput coli and thus gave rise to the erroneous impression. The operator drew the intestine down as far as possible into the left iliac fossa, where it was fastened, opened, and its contents evacuated. The child did well, the toxic symptoms manifested in the dry, yellow skin, anemia, and poor nutrition gave way, and at the end of six weeks the child had gained 12 pounds in weight. For four months evacuation was through the artificial opening, then as the child had made such great improvement, it was decided to resect the intestine. This was done at the Children's Hospital in San Francisco. Under ether the redundant sigmoid and the intestine above it up to what appeared to be the normal first part of the transverse colon were resected. After removal this redundant loop was 15 inches in length and in diameter about 3 inches, and its walls about 5/16 of an inch in thickness. The freed upper end was stitched into the severed end of the rectum; this was accomplished with great difficulty because the rectal tissues were thick from hypertrophy and the tube at that point was nearly three times as wide as was the implanted end of the large intestine.

The report of the microscopic section (Figs. 10 and 11) by Jean Oliver is as follows: "The sections show a normal mucous membrane with well-marked lymphoid follicles which are somewhat hyperplastic. All the muscular elements of the intestine are markedly hypertrophied, including the muscularis mucosae. The submucosa is rather fibrous and the submucosa and serosa normal. Many large dilated blood-vessels filled with blood are found."

The anastomosis was successful except that there was a small area of imperfect union which gave rise to a slight fecal fistula. This opening, however, closed spontaneously after about two weeks and the patient went on to apparent recovery with occasional attacks of retention of feces and moderate abdominal distention, which were apparently due to the fact that the huge rectum was deficient in tone and expulsive power. For some months the improvement was marked and progressive, but at no time has the patient done so well as during the period while he carried a freely opened artificial anus. The bowels have to be evacuated by the aid of small low enemas of glycerin or soap and water. This dislodges what appears to be a normal fecal mass from the rectum and each enema is followed by a normal-appearing evacuation.

Six months after the operation the mother stated by letter that some distention was recurring. This is undoubtedly due to the fact that the portion of transverse colon which was left suffered from the same pathologic defect as the portion amputated, although probably to a less degree, and this region is now giving way under the stress of pressure. It seems probable that the remaining colon will yet have to be removed.

CASE OF MICROCOLON WITH HYPERTROPHIED SMALL INTESTINE

During the period of our interest in megacolon Baby R., male, 5 days of age, was brought into the Lane Hospital *in extremis,* deeply jaundiced and cyanotic, incessantly vomiting, ejecting meconium-like material. The child had had no evacuation since birth, had taken but little food and had passed urine a few drops at a time only. The vomiting had persisted for forty-eight hours before entrance. The abdomen was distended and tense; it was possible to diagnose the presence of free fluid in the peritoneal cavity. The anal orifice was normal and the little finger moved freely into the rectum and was stopped just above the pelvic brim by a marked narrowing. A diagnosis of intestinal obstruction due to stenosis at the sigmoid was made, but the child was past aid and died an hour after admission. The necropsy was performed by Dr. William Ophüls, whose notes are as follows:

"Strongly built, male baby, 54 cm. in length. Cyanosis. Anus well formed, patulous. No obstruction in any of the natural orifices. Skin shows yellowish hue all over.

"Some gas in peritoneum and about 350 c.c. of distinctly fecal fluid. Peritoneum is inflamed and covered with soft greenish shreds of fibrin. Omentum is small, hyperemic, contains no fat. Transverse and descending colon very thin and contracted. Sigmoid flexure is pulled up out of pelvis to a certain extent and fastened by vascular adhesions to the mesentery of one of the loops of small intestine. These adhesions are below the point of greatest distention of the bowel. About 10 cm. above the ileocecal valve is a small perforation.

"Upper part of small bowel distended with gas, lower part very much distended; diameter of intestine below is 3.5 cm. in places. Further down distention decreases, increases again, and at the ileocecal valve the intestine has normal diamter.

"Above-described adhesion is below the point of greatest distention of bowel; about 10 cm. above the ileocecal valve is an oval perforation, about 5 mm. in diameter, situated close to the mesenteric attachment, through which fecal matter escapes.

"Upper part of the small bowel is filled with soft greenish brown fecal material. In lowest part of small bowel is thick, pasty material. The ileocecal valve is open. The large intestine is very much contracted. It is almost empty but in some places there is a small amount of dried-up mucus. The rectum is of normal caliber, its mucous membrane hyperemic, with some blood in rectum. Appendix is normal. In region of perforation mucous membrane shows marked hyperemia and extensive hemorrhages, and there is some evidence of necrosis of the mucous membrane. Very much distended part of lower small intestine shows well-marked hypertrophy of muscular wall, being almost 4 mm. thick in places. Mucous membrane is distended, part much inflamed, superficial erosions in places. Necropsy findings revealed no other abnormalities."

Sections were made from the hypertrophied areas of the small intestine by Jean Oliver, whose report is here given.

"Section of the small intestine through its enlarged part shows marked hypertrophy of the muscular layers. This is especially prominent when one considers that there is also great dilatation, which tends to obscure the hypertrophy of the walls. The mucous membrane is extremely hyperemic and there are many large hemorrhages, especially in the submucosa. A considerable layer of mucus covers the epithelium. The serosa is also hyperemic and there is

beginning cellular infiltration and deposit of fibrin on its surface. The blood-vessels are large, dilated and filled with blood.

"Sections of the microcolon show the same hyperemia and hemorrhages in the mucous membranes but to a less degree. The submucosa is quite fibrous and also shows considerable diffuse hemorrhage. The muscular layers appear thickened, probably owing to the contraction of the intestine. The subserosa shows marked cellular infiltration and there is a heavy deposit of fibrin in the serosa."

The case of microcolon is included in these reports, not only because of its striking contrast to the megacolon cases, but also because there was also present a condition of hypertrophy of the small intestine (Fig. 4) in which the changes in the muscle and fibrous tissues parallel in every pathologic way the changes of the analogous tissues in the large intestine found in the cases of Hirschsprung's disease. It always seemed to us that the innate pathology underlying megacolon is akin to, if not identical with, the changes in tissue found in hypertrophic pyloric stenosis, especially in that form wherein there is general hypertrophy of the circular muscle of the stomach wall as well as of the ring muscle at the pylorus (Fig. 5). This case of microcolon with gigantism of the small intestine provides a true example of the same marked growth of muscle and hypertrophy of fibrous tissue with gigantism of vessels occurring in the small intestine.

Microphotographs of the sections of the three conditions have been made in the standard Pathological Laboratory by Jean Oliver (Figs. 6, 7, 8, 9, 10 and 11). A study of them will reveal how like the muscle affection and the marked hypertrophy in the mucosa connective tissue and vessels is in the three conditions. The photograph of the stomach from a patient with pyloric stenosis also is evidence for this point of view.

The consideration of our cases of megacolon gives some evidence on disputed points. Certain writers have denied the congenital origin of this condition. Our Cases 1 and 3 exhibited symptoms at birth and in Case 1 a typically developed giant colon was revealed by operation and necropsy. Cases have been reported in which giant colon was discovered in prematurely born children.

It has been questioned whether the sigmoid was narrow in all cases, and it has been asked if the true origin of the dilatation and hypertrophy was not an obstruction that blocked the passage with feces and allowed the work of the intestine and the stress on it to be followed by increased production of muscle and ballooning from the retained gas. Case 4 of this series, in which the gigantism included every por-

tion of the intestine to the anus, shows that the sigmoid narrowing is not present in every case. Case 2 is instructive in demonstrating the potential and emergent dangers to life of the condition, and with Case 4 causes us to resolve that in the future even the youngest child encountered with this trouble shall have the benefit of immediate colotomy and an artificial anus. The well-being of Patient 4 during the time he carried an artificial anus was so striking and the operation is so easy that these facts, taken with the well-known tolerance of infants to abdominal interference, make such an operative interference most reasonable, especially when we consider the unhappy course of Case 2 which demonstrated so completely the extreme danger of temporizing and of trying to evacuate the bowel through the rectum in those cases of megacolon with a narrow sigmoid.

As one considers the condition of gigantism in the intestine, the question presents itself: Are there mild cases of the disease? One who deals much with infants cannot but feel that such cases occur, and are not very unusual. There are obstinately constipated infants, who, while not rachitic, have abdomens that bulge inordinately and in whom the bulging for the most part follows the line of the colon and fills out the flanks to a marked degree. Such children are apt to be uncomfortable, to take food badly and to fail in weight, but they are not distressed by flatus. When their bowels are kept free all the symptoms disappear except the ballooning of the large intestine, and growth seems to adapt the children in time even to this visceral enlargement, for the individuals in this group seemed to do well once they are past infancy. The final proof of this contention must rest on necropsy of such a child who has met death from another cause, as the condition itself is never fatal, nor indeed is it severe. Hertz has called attention to a class of infants in whom roentgenograms show a lengthened sigmoid, and it may very well be that such infants correspond in the group of megacolon to the cases of mild hypertrophy with spasm that we encounter in the pyloric stenosis group.

The proper feeding of infants with congenital enlargement of the colon is of importance and presents difficulties. High fat formulas are not adapted to this condition, as soap stools give more trouble in all classes of megacolon than do other types of stools. Giant colon cases seem to respond to high percentages of dextrin, yet care must be exercised, as fermentation and gas formation have to be discouraged. Enemas are on the whole the best means of producing bowel evacua-

tions. Castor oil is to be shunned, as is magnesia. In older children milk should not be given.

The clinical picture of congenital enlargement of intestine and the colon is so characteristic that it seems hardly needful to discuss the differential diagnosis. The one case in our series which led to confusion was that of the new-born infant. The differentiation between multiple stenosis and megacolon in the new-born is certainly difficult, but the character of the peristalsis and the mildness of the digestive smyptoms should have been a guide to a proper diagnosis. This case was rendered more difficult by the vomiting, which was extreme, and which probably is a most unusual accompaniment of congenital enlargement of the intestine. The only other condition which might possibly lead to a mistake in diagnosis is tuberculous peritonitis, and this mistake will never be made if the child is examined under an anesthetic by the bimanual method, a finger of one hand in the rectum and the other hand on the abdominal wall. The characteristic smooth feel of the healthy peritoneum instantly excludes the presence of tuberculous inflammation. The gristly fibrous bands and the rolled-up lower edge of the omentum in tuberculous peritonitis give to the examining finger a sensation that is absolutely pathognomonic. (In passing, it may be said that there is no maneuver in diagnostic examination of children of more value than this bimanual rectal examination if it be made while the child is under the influence of an anesthetic.) It is unlikely that any of the abdominal tumors will be confused and hardly possible that the dilatation due to chronic intestinal indigestion in rickety children will mislead one.

It is probable that megacolon is a more common deformity than is generally thought. The symptoms are present from birth. They are: an enlarged abdomen, visible intestinal peristalsis, obstinate constipation, later, anemia and toxemia from fecal retention, accompanied sometimes by recurring attacks of intestinal obstruction. Pathologic hypertrophy with dilatation may include the entire intestine, or the dilatation may cease at the sigmoid, which will be hypertrophied, but with a narrow lumen. That isolated segments of the large intestine may be affected while most of the intestine remains intact and healthy is to be doubted. Fatal collapse can occur from fecal obstruction, and such obstruction may be brought on by injudicious attempts to empty the bowel. The one measure that promises relief is operative procedure, which may be utilized in either two or three stages: colotomy,

with artificial anus (this cannot be done too early in the child's life or in the course of the case), later excision of the entire large intestine with anastomosis from the ileocecal region into the rectum; or anastomosis may be done as a second step and resection of the entire colon be made a third operation.

240 Stockton Street—350 Post Street.

ABDOMINAL SYMPTOMS IN PLEURISY AND PNEUMONIA

JOHN DORNING, M.D.
NEW YORK

The diagnosis of pleurisy or pneumonia is, ordinarily, a comparatively easy matter. Not infrequently, however, particularly in children, the early manifestations of a pleurisy or a pneumonia may be very indefinite, or they may so closely resemble those of appendicitis, or some other acute abdominal lesion, that sometimes it may be difficult, if not impossible, to avoid an error in diagnosis during the first few days of the disease.

My only excuse for adding to the somewhat extensive literature of this subject is to emphasize the importance of making careful or repeated examinations of the chest in these more or less perplexing cases in order to escape, if possible, the unpleasant consequences of injudicious surgical haste on the one hand, or the disaster likely to follow the delay of a necessary operation, through overcautiousness, on the other.

There are on record a number of cases of pneumonia in which an operation was done on the erroneous diagnosis of appendicitis. Most of us have seen cases of supposed ether pneumonia, detected after operation for an alleged mild catarrhal appendicitis, in which we were not fully convinced that the pulmonary inflammation was not the primary, if not the sole trouble. Nevertheless, a pneumonia may, though rarely, occur coincidentally with an appendicitis, both being caused by the same pneumococcus infection, or the one may follow the other.

During the past three years I have seen sixteen cases of pneumonia and pleurisy in which there were abdominal symptoms, such as vomiting, pain, tenderness, muscular rigidity and tympanitic distention, so pronounced as to call for more than ordinary care and sometimes some delay in reaching a correct diagnosis. In one case an operation had been performed, the history of which, though previously published, is again here briefly presented:

On Feb. 7, 1912, F. H., female, age 4 years, was taken with severe cramps in the lower right side of the abdomen. The medical attendant diagnosticated acute appendicitis and had her immediately admitted to the surgical division of St. Francis' Hospital for operation.

The record on admission notes a very sick child with a rigid and generally painful abdomen; too painfully tender to permit more than a superficial examination. Temperature 103 F., respirations 40, pulse 112. No cough. Family history negative. Child had whooping-cough when a year old; measles in her second year; no other illness.

Next morning condition unchanged. No cough. Temperature 103 F., respirations 40, pulse 118. Blood examination showed leukocytes 16,200, polynuclears 80 per cent., small lymphocytes 15 per cent., large lymphocytes 3 per cent., eosinophils 2 per cent. An exploratory laparotomy was performed but no pathologic condition was found in the abdomen. The appendix was not removed. The same evening the temperature was 104 F., pulse 130 and respirations 40. On the day after the operation the condition remained the same. No cough. A few moist râles were heard over both lungs which were thought to be due to the ether administered the day previous. A blood culture was made, with negative results.

On the third day the patient was transferred to the medical division. There it is recorded: Patient very sick, nervous and irritable; lips dry, tongue coated, breath offensive; tonsils enlarged; no exudate. Complains of pain on swallowing. No cough. Moist râles in chest have disappeared. Small area of dulness at base of left chest posteriorly. An empyema was suspected and an exploring needle was introduced without obtaining pus.

On the sixth day I was asked to examine the patient. Then the temperature was 104 F., respirations 40, and pulse 140. Respiration thoracic; no expiratory grunt; no cough. Palpation of the abdomen revealed only slight resistance, limited to the right side, and scarcely any tenderness. Over the lower third of the left chest posteriorly there was a moderate degree of dulness and a not well-defined bronchial respiration and distant bronchophony. Blood was taken for a culture.

Seventh day: Physical signs in the chest well-marked. Bronchial breathing in left chest very pronounced both anteriorly and posteriorly. No cough. The pathologist reported a pure and luxuriant growth of pneumococci in the culture which yielded positive reactions to agglutination tests. On the following day there were heard beginning signs of resolution in the left lung. The fever ranged between 100 and 103 F. until the end of the fifth week, and at the end of the seventh week she was discharged cured.

Here then was a case of pneumococcus septicemia in which the early symptoms were so misleading as reasonably to justify surgical interference.

The next three cases are selected as being fairly illustrative of the subject under consideration. The first one barely escaped operation, the signs of consolidation appearing on the third day. In the second a pulmonary inflammation was suspected because of the marked increase in the ratio of the respiration to the pulse rate, though a friction rub, the only evident physical sign, was not perceptible until

the fifth day. The third case presented no difficulty in diagnosis when admitted to the hospital, though it is more than likely that the abdominal symptoms occupied the attention of the attending physician to the exclusion of a careful search for trouble in the chest.

D. F. K., aged 3 years, was taken ill April 15, 1912, with vomiting and severe pain on right side of abdomen. Seen by the family physician the same evening. Temperature 102.6 F., respirations 35, pulse 120. Diagnosis of appendicitis and operation advised. I was called in the next morning to share the responsibility of an operation. At this time the temperature was 103 F., respirations 35, pulse 126. Respiration was thoracic with an occasional catch. No expiratory grunt. Complains of abdominal pain. Abdomen distended and rigid. Right iliac region so tender and painful as to permit only superficial palpation. Right thigh flexed. Examination of chest negative. Digital examination per rectum elicited no additional pain. Advised delay of operation, the responsibility of which I had wholly to assume.

On the following day the condition remained unchanged. Leukocytes, 14,000; polynuclears, 85 per cent. A blood culture was later reported negative.

The fourth day showed no amelioration of the abdominal symptoms and no change in the temperature, respiration and pulse. There was occasional cough. At the base of the right lung posteriorly there was heard distant bronchophony with slight dulness.

On the fifth day the abdominal symptoms had greatly subsided and the physical signs in the chest were more pronounced.

M. W., aged 6 years, was admitted to the hospital April 11, 1914, with a history of having become ill the previous day with severe pain in the stomach, and vomiting. There had been no errors in her diet. Bowels constipated; no cough. Child seemed to be greatly prostrated. Temperature 103 F., respirations 54, pulse 150. Rigidity and tenderness of the whole abdominal wall were present, which slightly yielded on the left side to gentle pressure with the flat of the hand, but became more resistant and very painful in the right iliac region. Examination of the heart and lungs was negative. After a dose of castor oil and an enema had relieved the constipated bowel the child next day seemed more comfortable, but the abdominal signs remained the same and an examination of the chest was still negative. There was an occasional catch at the end of inspiration. Temperature 101.8 F., respirations 46, pulse 135. Leukocytes 14,000.

On the following day, the fourth day of her illness, the tenderness and rigidity on the left side of the abdomen had subsided, but persisted in the right iliac region. Temperature 102.4 F., respirations 38, pulse 120.

Fifth day, the right-sided rigidity and iliac tenderness were less marked. The child complained of pain under the right scapula. Coughed a little for the first time. A distinct friction rub was heard over the posterior lower borders of both lungs and a few scattered moist râles over both sides of chest. Temperature 102.8 F., respirations 46, pulse 120.

Sixth day, the patient was bright and lively, no complaint of pain, abdomen soft and free from tenderness. Temperature 99 F., respirations 20, pulse 90. Friction rub had disappeared. Roentgen-ray showed two small shadows in the lower third of right chest.

C. B., aged 2 years, was sent into the hospital for operation after having been sick three days with a supposed appendicitis. On admission the temperature was 103 F., respirations 50 and pulse 120. No cough, no catch on inspiration or expiratory grunt. There were marked pain and tenderness over the right side of the abdomen, increasing in intensity towards the base of the chest, with moderate muscular rigidity that partly yielded to firm, gentle pressure with the flat of the hand. Examination of the chest revealed moderate dulness over the upper and posterior part of the right lung with high pitched respiration and some bronchophony. The child was given a dose of castor oil.

By the following afternoon the abdominal symptoms had subsided and the physical signs of pneumonia were more pronounced.

It has long been well known that pain may be a most misleading symptom for the reason that it is not always located at the seat of the disease causing it. For instance, the diagnosis of vertebral caries is sometimes delayed because of a misinterpretation of the referred epigastric pain, and disease of the hip joint is not always at once recognized on account of the complaint of pain in the knee. In some rare cases of renal calculus the pain may be felt in the opposite lumbar region, or the distressing symptoms may be located in the region of the bladder or at the head of the penis. Laennec, in alluding to the pain in pleurisy, remarks that occasionally from the beginning of the disease, we have a stitch on the right side and pleurisy on the left. Huss, in dealing with the subject, explains the pain on the opposite side by an occasional anastomosis of the right and left intercostal nerves.

The pain in pleurisy or pneumonia may be referred to almost any part of the abdominal cavity or it may be distributed over its whole area; most frequently it is limited to the right side or to the epigastric region and when associated with considerable local tenderness and marked rigidity of the abdominal walls and an absence of noticeable respiratory symptoms the attention may become so riveted on the painful region that only a superficial examination of the chest is made or the possibility of a pulmonary inflammation is entirely disregarded.

It is believed that an explanation of these painful abdominal symptoms may be found in an irritation, or in an actual inflammation, of one or more of the lower six intercostal nerves. These intercostal nerves are distributed to the lower part of the pleura, to the diaphragm and to the skin and muscles of the abdominal and thoracic walls. Irritation of the seventh to the ninth intercostal nerves would cause painful symptoms in the upper abdominal zone, while in involvement of the tenth and eleventh nerves there would be referred pain

and muscular rigidity in the lower part of the abdomen, producing symptoms resembling those of appendicitis.

This intimate nerve connection between the pleura, diaphragm and structures of the thoracic and abdominal parietes would seem to indicate a closely related function of these parts in connection with the respiratory movements. Hence the effort on the part of the intercostal and abdominal muscles, by their inactivity and rigidity, to restrict the movements of the chest walls and thereby lessen the pain and limit the damage to the underlying inflamed tissues.

The character of the pain is not always a reliable guide in the differential diagnosis, especially in children. The sharp, stabbing pain in the upper waist line, usually so expressive of a pleurisy, may sometimes be due to a peritoneal inflammation; and the colicky, or dull aching pain typical of a peritoneal irritation may be caused by a diaphragmatic pleurisy. This was strikingly exemplified in one case in which a diagnosis of a probable perinephritic abscess was made because of a severe, continuous dull pain, exquisite tenderness and muscular rigidity in the right upper third of the abdomen and negative chest findings for six days. It proved to be an acute pleurisy, the pain abruptly ceasing with the effusion of fluid into the pleural cavity.

This sudden relief of pain is rather diagnostic of acute pleurisy, indicating the separation of the inflamed pleural surfaces by an exudate or an effusion. It may, however, also be observed in an acute appendicitis when a much dreaded rupture or gangrene alleviates the tension in the inflamed part.

Superficial tenderness and rigidity of the abdominal parietes becoming intensified by a light touch, but which diminishes or disappears under gentle and firm pressure with the flat of the hand, would undoubtedly point to a pleurisy or a pneumonia rather than to an appendicitis. In one-third of the cases, however, there was no abatement of these symptoms on pressure, hence in such an event a pulmonary involvement cannot be positively excluded.

Relaxation of the rigid abdominal wall between respirations though generally regarded as an important diagnostic sign in pneumonia, was only occasionally noted.

A rapid catching respiration accompanied by an expiratory grunt, often heard on entering the sick-room, are so strongly characteristic of pleuropneumonia that the real nature of the trouble may be suspected or a correct diagnosis may even be made before the patient is

examined. They may also be observed in acute pericarditis. In two cases of acute suppurative appendicitis, in which there were at no time any discernible physical signs in the chest, these symptoms, to which was added a disproportion in the ratio of the respirations to the pulse and temperature (respirations 48 and 60, pulse 128 and 114 and temperature 102.5 and 102.8 F., respectively) were so pronounced as to excite a suspicion of a complicating diaphragmatic pleurisy.

Acceleration of the respiration, out of proportion to the pulse rate and fever, would be in itself very significant of a pneumonia; but it was not the invariable rule in these cases.

There was absence of cough during the first three days in eight cases; no cough until the fifth day in two; and none at all during the whole course of the disease in two instances, in one of which there were never any perceivable chest signs, though a crisis occurred on the fifth day.

The behavior of the temperature is not always a reliable guide, for the sudden elevation and continued high range, so common a feature of pneumonia, and usually considered an important differential symptom, is often seen in acute appendicitis. We also know that some exceptional and grave cases of pneumonia or peritonitis are attended with little or no fever.

Blood examinations gave varying results, the leukocytes ranging from 10,000 to 19,000 and the polynuclears from 70 to 90 per cent. In three cases a blood culture showed a pneumococcus, in one of them before the physical signs became manifest.

Digital examination per rectum was sometimes of material assistance in eliminating appendicitis. If gentleness be used the index finger can, without much discomfort to the patient, be carried up far enough in young subjects to detect tenderness or exudate in the region of the appendix.

After the first day or two the Roentgen ray will sometimes clear up the uncertainty attending the diagnosis of a central pneumonia. In private practice, however, the Roentgen ray is perhaps not likely to be available when most desired.

After all, it must rest with the attendant, after due consideration of all of the symptoms, whether it would be wiser to incur the risk of an exploratory operation in certain doubtful cases presenting urgent symptoms than to defer too long surgical interference for a possible ruptured appendix or some other serious abdominal condition.

A RESPIRATION INCUBATOR FOR THE STUDY OF THE ENERGY METABOLISM OF INFANTS *

JOHN R. MURLIN, PH.D.
NEW YORK

For the study of the metabolism of infants, especially new-born or premature infants, it is important to have perfect control of the temperature to which the child is exposed. Preliminary work on animals with a respiration chamber surrounded by water, the latter kept warm by means of a gas-flame placed underneath, convinced me that a more exact control was necessary before observations on the infant could be undertaken. In casting about for a satisfactory type of thermoregulator, the Freas oven came to mind and after conference with the inventor (Prof. Thomas B. Freas, Dept. of Chemistry, Columbia University) as to the feasibility of the application of its regulator to the purposes in hand, plans were agreed on for the construction of an electric incubator which would be suitable for the introduction of a respiration chamber large enough to contain an infant one year of age. Dr. Freas has placed the laboratory under special obligations for the interest he has shown in the execution of these plans.

This particular incubator measures 60 by 82 by 115 cm. outside and 46 by 48 by 100 cm. inside. It rests on a special iron stand provided with it, 85 cm. above the floor and makes a good appearance as a piece of laboratory or hospital furniture (Figs. 1 and 2).

The regular plan of construction of the Freas air thermostat[1] was departed from in only a few particulars. For example, it was necessary to have a considerable number of openings through the wall for the passage of the ventilating-pipes and cooling-coil, the introduction of thermometers, the connection of the oxygen regulator, etc. The range of temperature was fixed at from 20 to 50 C., the capacity of the heater being limited to 200 watts. The motor for driving the stirring-fan (Fig. 5, *F*) which is provided with the incubator was

* From the Physiological Laboratory of the Cornell University Medical College, New York City.

1. Freas, Thomas Bruce: A Study of Thermostats, Dissertation, University of Chicago, 1911.

mounted on a large steel spring so that the vibrations should be damped out to some extent. The glass doors were made as large as the front end, thus permitting plenty of light to enter and were provided with strips of felt so as to make the closure practically air-tight.

THE RESPIRATION CHAMBER

The chamber measures 30 by 32 by 76 cm. inside. It was made of 14-ounce tinned copper, strongly lock-jointed and soldered, and was rimmed at the front end by a heavy brass casting. Another casting of

Fig. 1.—General view of room in Bellevue Hospital containing respiration incubator (Eimer and Amend) set up ready for use. The inner chamber is shown through the glass doors of the incubator. Inside the chamber the bed is seen. The balances on the left of the room are used in weighing the absorbers. The balance in the middle of the room (sensitive to 1 gm.) is used in weighing the infant. Further description in the text.

the same dimensions was made as a frame for the glass head or door of the chamber. The glass is held in place by litharge cement covered with heavy wax and a coat of white-lead. A heavy rubber gasket is cemented to the brass rim, and a perfectly air-tight closure of the chamber is secured by clamping the frame of the head firmly against this gasket by means of twelve L-shaped steel bolts, which engage the

back side of the brass rim (Figs. 2 and 3). The chamber is mounted on wooden runners and is strongly reinforced by means of three galvanized iron ribs girthing it at equal intervals from front to back. Pressure changes inside or outside the chamber, therefore, do not alter its volume.

Since the incubator measures 46 by 48 by 100 cm. inside, a space 7.5 cm. wide is left on all sides of the chamber. The air in this space is kept in motion by a fan (Figs. 4 and 5).

Fig. 2.—Another view of the same room. The absorber table is seen at the right. The glass doors are open and the glass head of the inner chamber is lying on the shelf below the incubator. The stethoscope tubes may be seen lying on the nurse's table and the bell of the stethoscope inside the incubator. Further description in the text.

VENTILATION AND MEASUREMENT OF RESPIRATORY EXCHANGE

The ventilation of the respiration chamber is accomplished by means of a Benedict "universal respiration apparatus,"[2] connected to

2. Benedict, F. G.: Am. Jour. Physiol., 1909, xxiv, p. 345; Deutsch. Arch. f. klin. Med., 1912, cvii, 156; AM. JOUR. DIS. CHILD., 1914, viii, 1.

it by means of wide rubber tubing and ½-inch brass pipes. The brass pipes are screwed into strong brass bushings soldered to the copper wall. The inlet pipe enters at the upper back corner of the chamber on the right side and the outlet is connected with the lower front corner on the left. Thermometers are admitted to the inlet and outlet pipes by means of T joints (Fig. 3, *T*4). The arrows in Figure 2 show the direction of the air current.

The Absorber System.—The Benedict apparatus was so constructed for use with the incubator that by turning valves (V and V′, Fig. 2) the absorbers could be quickly changed after stopping the

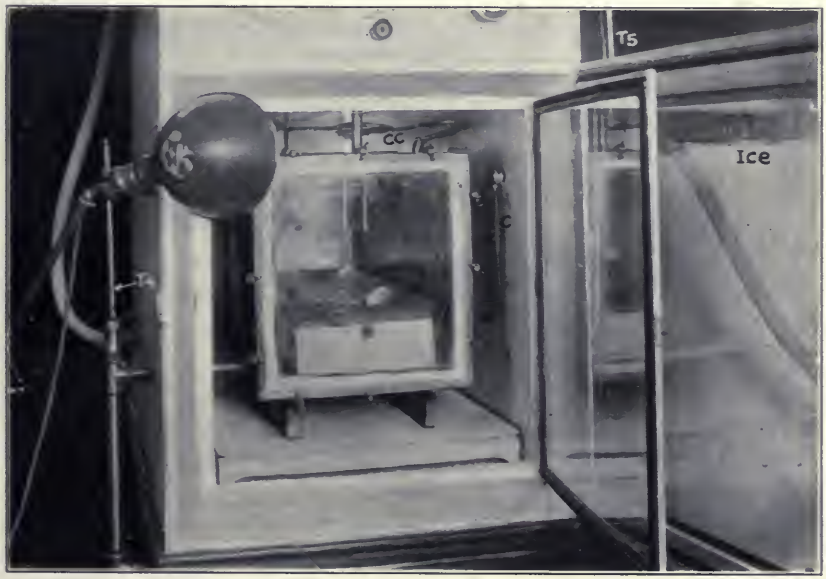


Fig. 3.—A near view of the interior of the incubator showing the cooling coil, *c.c.*

blower, without the loss of more than 20 c.c. of air.† Each set of absorbers consists of two soda-lime jars for absorption of carbon dioxid and a sulphuric acid absorber for catching the water lost from the soda-lime. Each of these absorbers is small enough to be weighed (separately) on a Sartorius balance sensitive to 0.001 gm. The soda-

† Since this was written the table has been reconstructed so two sets of absorbers can be connected at once and the change from one to the other accomplished with a single lever operating both valves.

lime absorbers are made on the plan (Fig. 1, *S. L.*) adopted for the absorbers used with the Sage calorimeter in Bellevue Hospital.[3] The sulphuric acid absorber is the gas washing-bottle designed by Williams[4] for use with the animal calorimeter in this laboratory. Immediately following the sulphuric acid absorber is a brass tower containing sponges kept moist with very dilute sodium hydroxid solution, which serves the double purpose of imparting a slight amount of moisture to the air and of arresting any traces of acid fumes which may come from the drying-bottle.

Since the water given off from the lungs of the child does not enter into the computation of the heat production by the indirect method (except as this water may affect the volume of the oxygen admitted), the absorbers used in the collection of this water are not weighed. They consist of two Williams bottles placed in series after the blower on the under part of the absorber table (Fig. 2, $H_2\ SO_4$). A brass trap (Fig. 2, *Tr.*), of a capacity about equal to the amount of the sulphuric acid contained in the first Williams bottle, is introduced for the safety of the blower. A bicycle coaster brake on the large pulley of the blower makes it impossible to turn the blower the wrong way; but the trap would catch any acid which might be drawn back by sudden blockade of the air-circuit at any point.

The Residual System.—The measurement of oxygen absorbed by the child is based on the general proposition that oxygen be admitted to the air-circuit just as rapidly as it is used. This presupposes that the respiratory products should be absorbed and removed from the air as rapidly as they are produced. The efficiency of the absorbers is such that as far as the welfare of the child is concerned the ventilation is perfect. Neither carbon dioxid nor water vapor can accumulate to any appreciable extent. But an *absolutely* perfect adjustment of the rate of ventilation so as to leave the composition of the air *exactly* the same at the end of a period as at the beginning is impossible, especially if the child should cry or void urine near the end of the period. Since the admission of oxygen is regulated by the total volume of the air, (see below) and since this volume will vary with the amount of water vapor in it as well as with the temperature and barometric pressure, it is necessary to know the composition of the air at the close as well

3. Lusk, Soderstrom and Riche: Arch. Int. Med., to appear soon.
4. Williams: Jour. Biol. Chem., 1912, xii, 323.

as at the beginning of a period. The analysis is carried out by diverting a measured sample of air from the main circuit, causing it to flow through a special set of absorbers and returning it to the system without loss of anything more than the small quantities of carbon dioxid and water contained in it. These analyses are called the residuals because they give information as to the amounts of the various gases residual in the air at definite moments.

A small copper pipe is tapped into the main circuit on the positive side of the blower (Fig. 2, *R*) and is led upward through the top of the table. By turning a stop-cock at the base of this pipe a small fraction of the total air current can be drawn off. The residual absorbers consisting of three U tubes (Fig. 1, *R*) (the first containing sulphuric acid and pumice stone, the second finely granular soda-lime and the third sulphuric acid and pumice stone again)[5] are connected to this small pipe and the air having passed through them is conveyed to the meter (Fig. 2, M) where it is measured, thence through a drying-tower[6] (the meter contains water) and back to a point (Fig. 2, x) in the main system where the pressure is low.

The U tubes are weighed to the fourth decimal place before and after drawing off the sample. When the amounts of water vapor and carbon dioxid in this measured sample (usually 5 liters) are known, the total amounts in the entire system are readily calculated. By a series of corrections which will be better understood after the method of conducting an alcohol check is explained, the amount of oxygen residual in the air at the time the air sample is taken can also be computed.

The Oxygen System.—Oxygen is admitted into the ingoing air so that it may be thoroughly diffused into the chamber. The amount admitted is regulated by the method first described by Benedict and Carpenter[5] and as slightly modified by Williams[4] in his construction of the animal calorimeter. The essential parts of this arrangement consist of a rubber bag or spirometer which serves as a tension equalizer and volume indicator at the same time (Figs. 1 and 2, *RB*) and a magnet (*Mag.*), which, when actuated, releases pressure on a rubber tube leading from the oxygen cylinder (O_2). The rubber bag

5. Cf. Benedict and Carpenter: Carnegie Institution of Washington, Publ. 123, p. 73.

6. The drying-tower is not shown in the figure because it stands behind the meter.

communicates with the respiration chamber by means of a brass pipe, and a valve in this pipe permits the bag to be shut off from the chamber. The oxygen cylinder[7] while in use is suspended from a strong hook at the right side of the apparatus conveniently near the magnet. The magnet is connected through a resistance board carrying three lamps and through the mercury cups (*C*), situated on the horizontal arm above the magnet, with the house current. A lever (*L*) 38 cm. long is pivoted above the bag and is so counterpoised that it rests lightly on the bag by means of a broad metal disk. The arm ends at the right in a point which traverses a scale (*Sc*). It carries contact points which dip into the mercury as the lever descends. As the oxygen in the air of the circuit is used up by the child the bag collapses until the lever makes contact with the mercury (sparking is prevented by means of a condenser not shown in the figures). This actuates the magnet and releases pressure on the pure gum tubing leading from the oxygen cylinder. Oxygen thereupon enters the circuit until the volume is increased sufficiently to break the electric circuit, when pressure is again applied to the tubing thereby shutting off the oxygen; and so on.

At the beginning of a period oxygen is let into the system by hand until the pointer of the lever rests at a certain mark on the scale. At the end of the period the system is again filled to exactly the same point. If the volume of the air has not changed (by change of temperature, pressure, or composition of the air) the loss in weight of the cylinder would be the amount of oxygen consumed. Such conditions, however, would be very exceptional; hence all corrections must be made for every period.

Oxygen cannot be let into the system by any such automatic arrangement until the pressure which exists in the cylinder is reduced to a pressure which the rubber tube will withstand. A valve which reduces this pressure to a few pounds per square inch was made for these cylinders by the mechanician of the laboratory, Mr. James Evenden. It is shown in Figure 4.

Switch-Board.—A special switch-board, the end of which can be seen in Figure 6, provides electric outlets for: (1) the heater of the

7. Purchased from the Consolidated Dental Mfg. Co., New York. These cylinders measure 4 cm. diameter by 33 cm. in length. They will contain about 50 gm. of oxygen under a pressure of 100 atmospheres. Together with the reduction valve the cylinder weighs about 1.5 kg. and can be weighed on the Sartorius balance to 0.001 gm.

incubator; (2) the fan motor; (3) the motor on the absorber table; (4) the oxygen regulating system, and (5) any extra lamps required for illumination of the balances (Fig. 1), the interior of the incubator (Fig. 3), etc.

The Cooling-System.—The walls of the incubator being constructed of materials having a low coefficient of conductivity, the heat

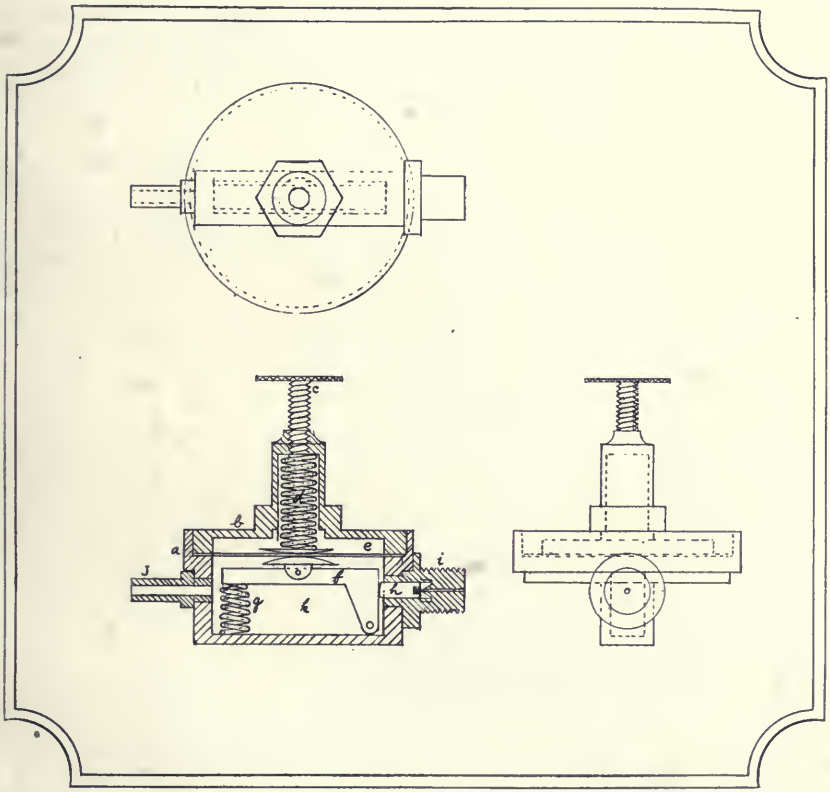


Fig. 4.—Detail of reducing valve used on small oxygen cylinder: *A*, case; *B*, lid of case; *C*, screw to compress spring; *D*, spring to reduce diaphragm; *E*, diaphragm; *F*, lever to close valve; *G*, spring to raise lever; *H*, valve with hard rubber tip to control oxygen; *I*, end to screw into oxygen cylinder; *J*, end for hose, oxygen supply. Oxygen contained in cylinder is held back by valve *H*. By compressing spring *D* with screw *C* lever *F* is lowered, thus allowing valve to come back and permitting oxygen to enter chamber *K* until the pressure beneath the diaphragm is sufficient to bring it to its original position which shuts off the oxygen again. When the oxygen in chamber *K* is below the desired pressure the tension of spring *D* again lowers the lever and repeats the operation.

produced by the infant would not be conducted away as rapidly as produced and the temperature inside would rise unless the room air were at a very low temperature. To counteract this tendency and to furnish a chilling effect against which the heater of the incubator should operate, a cooling-system was installed. This consists of a coil (Fig. 3, *cc*) of copper pipe, ¼ inch inside diameter, making several turns on the two sides and the top of the inner wall of the incubator and carrying a stream of tap-water. At ordinary room temperature and with tap-water coming directly from the mains (that is, not warmed unduly

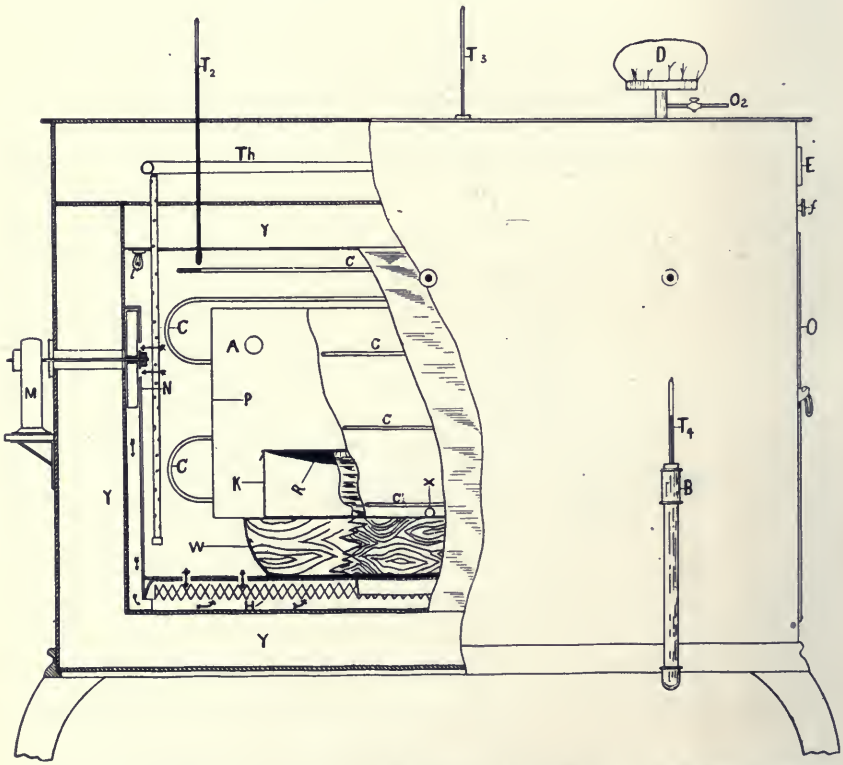


Fig. 5.—Sectional view of the incubator from the side. *Y*, asbestos packed wall; *H*, heater coil; *C*, cooling pipes; *P*, copper wall of inner chamber; *A*, point of entrance for air; *B*, exit pipe for air, containing thermometer; *W*, wooden runner which supports copper chamber; *K*, bed inside respiration chamber; *R*, rubber diaphragm; *Th*, thermo regulator; *M*, fan motor; *N*, fan. Arrows show direction of currents which equalize temperature inside the incubator. *D*, rubber bag for regulation of oxygen; *l*, lamp; *f*, control for setting regulator at any desired temperature.

by the house pipes) it is not necessary to chill the stream. It flows, however, through a coil of pipe inside an ice-tank (Figs. 1, 2 and 3), and in very warm weather the water can easily be kept down to the desired temperature by placing ice in contact with the coil. To secure a constant flow the water is given a constant head of pressure by passing it through a flush tank, operated by a ball cock, placed near the ceiling of the room. (This tank is too high to be seen in any of the figures, but the pipes leading to and from it are seen.) After passing through the cooling-system the water is conducted back to the sink.

A thermometer (Figs. 1 and 3, *T*5) is placed in the stream of water, and a wheel valve is placed near the tank, so that the cooling effect can be regulated to some extent both by the amount of ice and by the rate of flow of the water. The thermoregulator of the incubator takes care of the finer regulation. The heater is sufficient to keep the space about the respiration chamber at 27 C., even when the water is at 4 C. and the rate of flow 400 to 600 c.c. per minute. This would be wasteful of electric current, however, and a water temperature of from 10 to 15 C. with a rate of 200 to 300 c.c. of water is more than sufficient to carry away the heat produced by a year-old infant even in very warm weather, unless the infant cries actively. In the latter case the speed of the water can be increased, or more ice can be introduced or the heater can be turned off. With such a cooling effect as just mentioned the temperature outside the chamber falls very quickly, and within a very few minutes also the temperature inside the chamber.

HEAT CONTROLS

Many control tests were made with the incubator as adapted in this way to the purposes of a respiration apparatus, both empty and with animals, before it was used with infants. Four thermometers are regularly used in the following positions: No. 1 (not shown in any illustration) in the ingoing air; No. 2 on the top at the rear end of the incubator, the bulb extending into the outer air space; No. 3 (T_3, Figs. 1, 2 and 5) on top near the center, the bulb extending 1 inch into the inner air-space, and No. 4 in the outgoing air. All these thermometers are graduated to tenths of a degree C. and can be read to 0.05°. An air thermometer is usually kept on the wall near the absorber table.

Table 1 will serve as an example of the accuracy of the heat control. The subject was a small black dog, 4 kg. in weight, which lay perfectly still the entire hour.

TABLE 1.—HEAT CONTROL SUBJECT, SMALL BLACK DOG

	Temperature			
Time P. M.	Thermometer No. 1 Ingoing Air Degrees	Thermometer No. 2 Between Walls Degrees	Thermometer No. 3 Inner Chamber Near Center Degrees	Thermometer No. 4 Outgoing Air Degrees
1:50	23.0	27.0	28.1	27.9
1:55	23.1	27.2	28.1	28.0
2:00	23.1	27.2	28.1	28.1
2:05	23.2	27.3	28.1	28.1
2:10	23.3	27.2	28.1	28.1
2:15	23.4	27.2	28.15	28.1
2:20	23.5	27.1	28.15	28.1
2:25	23.5	27.15	28.15	28.1
2:30	23.5	27.15	28.15	28.1
2:35	23.5	27.1	28.1	28.15
2:40	23.5	27.2	28.1	28.1
2:45	23.6	27.0	28.1	28.1
2:50	23.6	27.0	28.1	28.1

The air temperature rose 0.6 of a degree during this observation and the temperature in the outer air space fluctuated somewhat according as the heat happened to be on or off at the moment of reading; but the temperature inside the inner chamber hardly varied at all during the entire hour.

CONTROL BY COMBUSTION OF ALCOHOL

The accuracy of the entire respiration apparatus has been checked many times in the course of the year and a half during which it has been in use, by combustion of alcohol in the chamber and the determination of the "respiratory quotient" of the flame. In the great majority of these check experiments all corrections have been made; but at times it has been sufficient to measure the oxygen by loss in weight of the cylinder with a correction for argon and the carbon dioxid by gain in weight of the absorbers without further corrections. With the flame burning uniformly the carbon dioxid residual in the circuit often does not change at all from the beginning to the end of a period. The water almost always falls the first period and then remains constant.

An example of a complete check experiment is given showing the manner of making all corrections. The same corrections and the same methods of calculation are used when the subject is a child.

Dec. 23, 1913. Alcohol check experiment.
Contents of respiration chamber — alcohol lamp and bed.
Vol. of air containing CO_2 67 l.
Vol. of air containing H_2O 66 l.
Vol. of air containing O_2 and N_2 69 l.
First period from 4:52 to 5:52 p. m.
At 4:52 residual analysis.
5 liters air contain 0.0178 gm. CO_2 and 0.0136 gm. H_2O
67 liters air contain 0.238 gm. CO_2 (=0.121 l.)
66 liters air contain 0.179 gm. H_2O (=0.223 l.)
To find residual O_2.
Temperature 28.06 C.; barometer 765.1 mm.

$$\log. 69 = 83885$$
0.121 l. CO_2 + 0.223 l. H_2O = 0.344 l.

$$\log. \frac{1}{1 + .00367(28.06)} = 95740$$

$$\log. \frac{765.1}{760} = 00290$$

79915 = 62.973 l. at 0° and 760 mm.
0.344 l. CO_2 and H_2O

62.629 l. O_2 and N_2
Substracting four-fifths for N_2 50.1032

12.5258 l. O_2

At 5:52 residual analysis.
5 liters air contain 0.0178 gm. CO_2 and .0136 gm. H_2O
67 liters air contain 0.238 gm. CO_2 (0.121 l.)
66 liters air contain 0.129 gm. H_2O (0.223 l.)
To find residual O_2.
Temperature 28.23 C.; barometer 765.3 mm.

$$\log 69 = 83885$$

$$\log. \frac{1}{1 + .00367(28.23)} = 95719$$

$$\log. \frac{765.3}{760} = 00302$$

79906 = 62.960 l. at 0° and 760 mm.
.344 l. CO_2 and H_2O

62.616 l. O_2 + N
Substracting N with which exp'm't started, 50.1032

12.5128 l. O_2

"Respiratory Exchange" Period 1
Gain in weight of CO_2 absorbers 4.850 gm.
Loss in weight of O_2 cylinder... 5.090 gm.
0.6 % correction for argon = 0.03 gm.

Loss in weight due to oxygen = 5.060 gm.

Residual analyses show: (1) no change in CO_2 of air but (2) amount of O_2 is less. (12.5258 — 12.5128 = .0130 l. = .0186 gm.) This shows that flame

used a little more O_2 than was admitted; therefore, 5.060 + .0186 = 5.079 gm. O_2 actually used. Reducing weights to volume:

log. weight CO_2 (4.850 gm.) = 68574
log. weight to vol. (CO_2) 70680

39254 = 2.469 l.

log. weight O_2 (5.079) = 70578
log. weight vol. (O_2) = 84496

55074 = 3.554 l.

$$\text{R. Q.} = \frac{\text{Vol. } CO_2}{\text{Vol. } O_2} = \frac{2.469}{3.554} = .695$$

Second period from 5:52 to 6:52.
At 6:52 residual analysis.
5 liters air contain 0.0166 gm. CO_2 and .0114 gm. H_2O
67 liters air contain 0.222 gm. CO_2 (= 0.113 l.)
66 liters air contain 0.150 gm. H_2O (= 0.187 l.)
0.113 + 0.187 = 0.300 l. CO_2 and H_2O.
Temperature 28.4 C. Barometer 765.3 mm.

$$\log. 69 = 83885$$

$$\log. \frac{1}{1 + 00367(28.4°)} = 95695$$

$$\log. \frac{765.3}{760} = 00302$$

79882 = 62.924 l. at 0° and 760 mm.
0.300 l. CO_2 and H_2O

62.624 l. O_2 and N
Subtracting N_2 with which exp'm't started 50.1032

12.5208 l. O_2

"Respiratory Exchange" second period.
Gain in weight of CO_2 absorbers 4.968 gm.
Residual analyses show that CO_2 in air was less at 6:52 than at 5:52 (0.238 — 0.222 = .016) 4.968 gm.
.016

Actual amount of CO_2 given off = 4.952 gm. = (2.521 l.)
Loss in weight of O_2 cylinder..... 5.500 gm.
Less 0.6 per cent. for argon..... .033

5.467 gm.

Residual analyses show more O_2 in air at 6:52 than at 5:52
12.5208 — 12.5128 = .0180 l. = .026 gm.
5.467
.026

Actual amount of O_2 used by flame = 5.441 gm. = (3.807 l.)

$$\text{R. Q.} = \frac{2.521}{3.807} = .662$$

First period... .695
Second period .662

Average... .677 Theory .666; error 1.6 per cent.

Table 2 gives the respiratory quotients obtained with the alcohol lamp for all the experiments between June 1, 1913, and July 1, 1914, on which all corrections could be made:

TABLE 2.—RESPIRATORY QUOTIENTS FOR EXPERIMENTS BETWEEN JUNE 1, 1913, AND JULY 1, 1914.

Date	Quotient	Date	Quotient	Date	Quotient	Date	Quotient
6/ 5/13	0.654	1/ 5/14	0.652	1/20/14	0.664	2/ 9/14	0.684
	0.675	1/ 8/14	0.642		0.656		0.644
	0.655		0.628				0.641
			0.667	Average...	0.660		
Average...	0.661			2/ 3/14	0.656	Average...	0.656
7/30/13	0.669	Average...	0.646		0.653	3/ 4/14	0.645
	0.670	1/11/14	0.661		0.644		0.637
			0.671				
Average...	0.669			Average...	0.651	Average...	0.641
12/ 1/13	0.617	Average...	0.666	2/ 5/14	0.673	6/24/14	0.627
	0.724	1/16/14	0.741		0.631		0.666
			0.610		0.644		
Average...	0.670		0.638			Average...	0.642
12/23/13	0.695			Average...	0.649	7/29/14	0.652
	0.662	Average...	0.663				
Average...	0.677						

It will be seen that a perfectly theoretical result not infrequently occurs in a single period, but that often one period shows a high and the succeeding one a low result, the mean being very near to the theoretical value. This in most instances is due to a rise or fall of temperature near the end of the first period. The average error for all of the daily averages is 1.8 per cent.

CONTROLS WITH DIABETIC DOGS

Dogs completely under the influence of phlorhizin or deprived of the pancreas have a total incapacity to oxidize glucose. This results in a respiratory quotient[8] which varies but slightly from 0.69. The diabetic dog, therefore, is a suitable subject for controlling the efficiency of a respiration apparatus of this type. The apparatus here described has been used in the study of certain problems in connection with pancreatic diabetes.[9] The following are examples of the quotients

8. With the depancreatized dog this level is not invariably reached until about the fourth or fifth day after operation. Cf. Verzár: Biochem. Ztschr., 1914, xxvi, 75.

9. Murlin, Edelmann and Kramer: Jour. Biol. Chem., 1913, xvi, 79 and Kramer and Murlin; ibid., 1914.

obtained on dogs which were known from the relation of dextrose to nitrogen in the urine to be completely diabetic:

Dog VI.	July	9, 1913.	R. Q.	11.27–12.27	.68
				12.27– 1.27	.68
Dog C.	July	23, 1914.	R. Q.	3.42– 4.27	.69
				4.27– 5.13	.68
				8.03– 8.49	.68
Dog E.	Aug.	4, 1914.	R. Q.	5.27– 6.27	.683
				8.27– 9.27	.685

Animal experiments of this kind furnish a better control of the efficiency of the apparatus for work with infants than does the alcohol flame, for the reason that both the chemical and the physical properties of the air are more nearly like those of an experiment with the child. As a matter of course, the entire air circuit is thoroughly sterilized by means of formaldehyd, circulated by means of the blower, before the apparatus is used with the infant.

OXYGEN BLANK

This form of control, introduced by Williams,[10] has been found useful as a check on the gas analysis. It consists in sealing up the chamber, starting the blower and taking off a sample of air just as in starting a period for an alcohol check, but without a subject or lamp in the chamber. If the air which is thus closed in the circuit is somewhat moist, the water absorbers will remove the water and its place will be taken by oxygen from the cylinder. The same would happen if the temperature were permitted to fall. At the end of an hour another sample of air is taken for analysis, and after the corrections for temperature and pressure are made, the amount of water lost from the air, as indicated by the residual analyses, should be equal to the amount of oxygen which has been lost from the cylinder. In such a check conducted on June 22, 1914, the amount of oxygen admitted was 0.346 liter in one hour. The air lost 0.411 liter of water; the carbon dioxid did not change. The temperature had risen 0.2° and the barometer had remained stationary. The temperature change caused an expansion of 0.056 liter in the entire system which would serve to keep out that much oxygen. Hence $(0.346 + .056) = .402$ l. of oxygen, with no temperature change, would have been admitted to replace a (calculated) shrinkage of 0.411 liter.

10. Williams: Jour. Biol. Chem., 1912, xii, 346.

REGISTRATION OF MUSCULAR ACTIVITY OF INFANT

The fan motor can be utilized for driving a kymograph situated on top of the incubator (Fig. 6). A small counter-shaft (*cs*) driven by a worm gear working on the axle of the fan carries a small pulley at its outer end and this connects directly with the driving pulley of the kymograph.

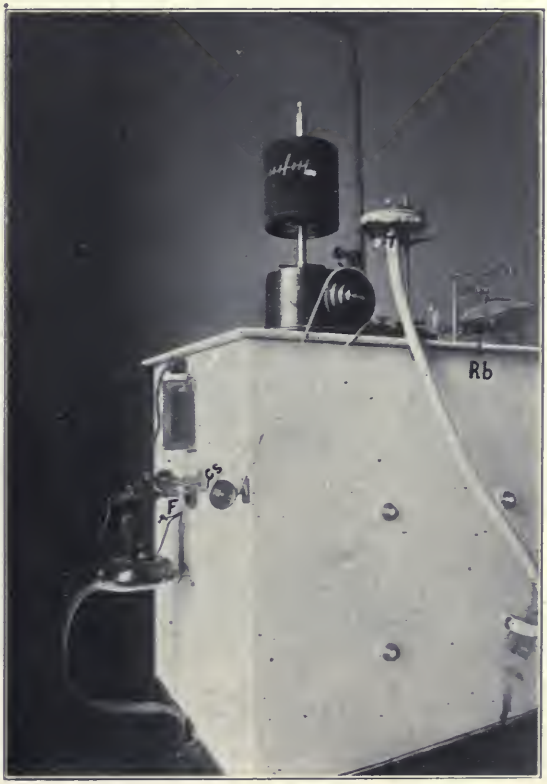


Fig. 6.—View of the rear end of the incubator showing fan motor (*F*); counter shaft (*CS*) for driving kymograph, and large recording tambour (*T*) for registering movements of the infant. *Rb*, rubber bag for equalizing tension and regulating flow of oxygen into the circuit.

For recording the actual motions of the infant several devices have been employed. One of them employed with new-born infants consists of two large tambours connected by wide rubber tubing (Fig. 7). One of these tambours is the infant's bed while in the respiration

chamber. The other (6 inches in diameter) carries a lever which writes directly on the smoked paper.

The bed consists of a galvanized iron box (10 by 22 by 61 cm.) covered with a piece of rubber sheeting. The rim of the box over which the sheeting fits is so made that the rubber comes in contact successively with two surfaces, each about 1 cm. broad, before it reaches the outside surface of the box itself. The corners, of course, are all rounded. An air-tight joint was secured by means of thick

Fig. 7.—Rubber bed used for recording motions of the child, showing the wide tube and large tambour necessary for quick transmission. The character of the tracings obtained is seen in the open book (A).

alcohol shellac. After the joint had been coated heavily with the shellac, the box was left for some days inverted on the rubber surface and weighted. Then the second surface was treated in the same way and the rubber tied firmly with a heavy cord. After several days' drying it was found to be air-tight. As a receiving plate for the infant's body a piece of linoleum of appropriate size was cemented to the upper surface of the rubber sheeting. A light blanket placed over this made a very comfortable bed.

Every motion of the infant was transmitted as an air wave to the recording tambour. In fact, the only objection to the device was its extreme sensitiveness. Even the pulsations of the air caused by the ventilating blower were recorded (Fig. 7 A). With older infants[11] the bed was discarded and the muscular motions were recorded by means of a cuff on the leg and a very flexible pneumograph placed about the chest.

11. See paper by Murlin and Hoobler.

PRIMARY PULMONARY ACTINOMYCOSIS IN A CHILD AGED TEN

WITH POST-MORTEM EXAMINATION

FRANCIS HUBER, M.D., AND SAMUEL BERKOWITZ, M.D.
NEW YORK

Actinomycosis pulmonis as a primary condition, though uncommon in America, has been observed rather frequently abroad. In the German literature Aschoff, Heuser, Rutinmeyer, Köster, and Kashiwanuva report such cases in detail, the latter describing four instances, while Lindt described a single case of primary involvement of the apices. In America, case reports of only a very limited number of this type have been recorded, owing perhaps, to a great extent, to the relative infrequency of the various types of this ailment. The disease may be acute or chronic, local or general. The symptoms vary within wide limits; they may be pronounced or, on the other hand, indefinite.

Actinomycosis, as we know, is an infectious disease caused by a specific micro-organism, *Actinomyces bovis,* originally named "ray fungus" by Bollinger and Hartz. It must not be confounded with the so-called pseudotuberculosis, streptothrix or cladothrix infections or atypical actinomycotic processes described by J. H. Wright under the name of "nocardioses."

It was first recognized in man by Langenbeck in 1845, who described a case of vertebral caries—the pus containing numerous "sulphur-like" granules. Twelve years later, in 1857, a patient with thoracic involvement was reported by Lebert. Murphy of Chicago, in 1883, was the first clinician on this side to report a patient suffering with this disease. The identity of the human and bovine disease was first pointed out by Ponfick. In a masterly monograph, Israel, in 1885, proved that the disease was transmissible from man to animal. The experiments proved that the infection could be transmitted from man to rabbits. These observations were later confirmed by Ponfick, Wolff, Hanan, John and Boström. Hodenpyl (1890) deserves the credit of having reported the first case of pulmonary actinomycosis in America.

The organism is widely distributed in nature and is easily isolated from certain grasses. Direct contagion is denied by many, though a few cases have been reported in which men have contracted it from animals. At the Royal Victoria Hospital one of the earliest cases occurred in the nurse attending a case of actinomycosis.

The infection may take place as follows: through (*a*) the mouth and pharynx, (*b*) the respiratory tract, (*c*) the gastro-intestinal tract, (*d*) the skin and wounds, and (*e*) idiopathically (?).

Pathologically, two types of lesions are recognized as characteristic of this disease, that is, (1) neoplastic and (2) inflammatory. Each type has selective tendencies in the special species chosen. Horses and cattle suffer more frequently from the former and man and hogs from the latter. The neoplastic type usually occurs in very vascular tissues and clinically resembles sarcomatous invasions, which may, however, result in spontaneous recovery. The inflammatory type which is most frequently seen in man, usually avoids parenchyma, spreads rapidly and forms characteristic sinuses from which the sulphur-like granules are discharged and can be shown microscopically to contain the "rays." Microscopically the actinomycotic area lends itself to division into three zones; from within outward they are: (*a*) the central zone of fungi and detritus, (*b*) the middle zone of round and epithelial cells which supplies the inner with detritus, and (*c*) the outer zone of granulation tissue.

The bacteriology is of especial interest to the laboratory worker, and although it is deserving of more than a passing remark, space does not permit any extensive references.[1]

The severity, location and type of the infection determines the nature of the symptoms. The sites are the following in their order of frequency: (1) cervical-facial, 55 per cent., (2) thoracic, 20 per cent., (3) abdominal, 20 per cent., of which more than half (50 to 60 per cent.) involve the cecum, and (4) cutaneous, 5 per cent. In the thoracic form definite signs and symptoms are usually absent until the disease is far advanced. Cough, with fetid sputum, consolidation and cavity formation may appear late, with attending cachexia, anemia and temperature of a variable type. The presence of an inflammatory swelling or a board-like induration of the thoracic wall is suggestive;

1. For a full description of the micro-organisms of actinomycosis, the reader is referred to Wright, J. H.: Biology of the Micro-organisms of Actinomycosis, Jour. Med. Research, new series, 1905, viii, 349.

later on suppurating foci or sinuses may result and arouse our suspicions as to the real nature of the process, which may be confirmed by a microscopic examination of the pus. Pulmonary cases, it has been asserted, are usually secondary to the abdominal variety. Because of the involvement of the right lower lobe in the majority of the cases it is thought that the lymphatic channels are to a great extent responsible for the transmission of the infection from the right side of the abdomen to this region. Infection through the blood-stream, however, seems more logical together with "locus resistentiae minoris" as in other infections, particularly so when the lung has been exposed to trauma.

The exact diagnosis can be readily established in many instances by microscopic recognition of the fungi from the sulphur-like bodies found in most cases. It is necessary that these granules be examined in thin preparations to avoid error—the liability "to look but to see not." Translucent bodies are positive evidences of such involvement, especially if club-shaped.

The prognosis, according to McKenty, varies with the extent, severity, and location—the nearer the surface or within the scope of radical surgical interference, the greater the possibility of early and successful cure. Therefore, it is evident, as this writer states, that cervical infections give a more favorable prognosis and are fatal in only 11 per cent. of cases, while on the other hand the abdominal mortality ranges as high as 71 per cent. and the thoracic even higher—83 per cent.

Treatment of actinomycosis is symptomatic and consists in the administration of potassium iodid continued over a long period of time. The drug appears, however, to act less favorably in the pulmonary type. The surgical treatment of this variety has not given brilliant results. The Roentgen ray is of doubtful value. Vaccine treatment has given some success after a considerable period.

CASE-REPORT

History.—J. B., a schoolboy, aged 10, of Italian parentage, was admitted to the Beth Israel Hospital (Dr. Huber's service), June 18, 1913, in a drowsy and restless condition, speaking only at times, always incoherently. The following history was obtained with difficulty through an interpreter. It was not until some time later that we learned that the boy was in the habit of spending much of his time in a stable.

Present Illness.—About four weeks before admission the child was struck on the chest by a man, with sufficient force to give rise to considerable soreness

of the right side and pain, causing him to complain for a few days. Two weeks later he was suddenly seized with high fever, headache, anorexia, prostration, and vomiting, the latter continuing for three days after the onset. On the fourth day of the illness an unproductive cough developed and was soon followed by a thin brownish expectoration which afterward became greenish and fetid. and was later accompanied by dyspnea. Three days before admission he began to show evidences of restlessness, drowsiness and incoherence, which have been increasing.

Previous History.—The previous history of the patient may be summarized in the following few remarks. "Instruments" were necessary at his birth. He was breast-fed until one year of age. His habits were always good. He was never sick before. The family history is without importance.

Fig. 1.—Pulmonary actinomycosis, showing extensive involvement of right side.

Physical Examination.—The patient appeared fairly well developed and nourished. Marked dyspnea, great prostration and extreme facial pallor were observed immediately on approaching his bed, together with a disagreeable fetid odor which pervaded that section of the ward. The skin was warm and dry. Eye examinations were negative. Extensive herpes labialis was present. The tongue was thick and coated, brownish white, and the pharnyx was congested. The teeth appeared normal.

Thorax: Since, in the history, the chief complaints centered in the respiratory apparatus, the thorax and its contents received particular attention. The movements of the right chest-wall were greatly diminished during both acts of respira-

tion. There was marked induration of the anterior and lateral aspects of the right side of the chest, particularly below. Though the regions involved felt hard, there was no evidence of local inflammation, no redness, no tenderness, increased local heat or deep-seated fluctuation. The interspaces were more prominent on the opposite side. Percussion over the affected side gave a "boardy" note which was diminished from above downward. The breath-sounds were very distant and bronchial in character, heard best after coughing. The voice and whisper sounds were also of this type. Fine and large moist râles accompanied the breath-sounds. These physical signs extended to the lower lobes but also diminished in intensity. Over the whole left lung there were areas of compensatory exaggeration of the normal findings together with scattered portions of bronchial breathing and fine moist râles.

Examination of the heart showed it to be in the normal position.

Abdomen: The right lobe of the liver was pushed down about an inch below the costal margin; otherwise the findings here were normal.

Course.—On admission to the hospital, the patient's temperature was 103.6 F. It continued remittent, ranging from 101 to 105, with a corresponding increase in the pulse-rate. The respirations varied from thirty to fifty per minute.

The blood examination showed 12,000 leukocytes with 84 per cent. polynuclears and 16 per cent. mononuclears (150 cells counted). Four days later a second blood count gave 9,200 leukocytes with 79 per cent. polynuclears and 21 per cent. mononuclears (the second count was made after the thoracotomy). Therefore, during a period of four days, the patient's infection was becoming exceedingly severe and his resistance poorer.

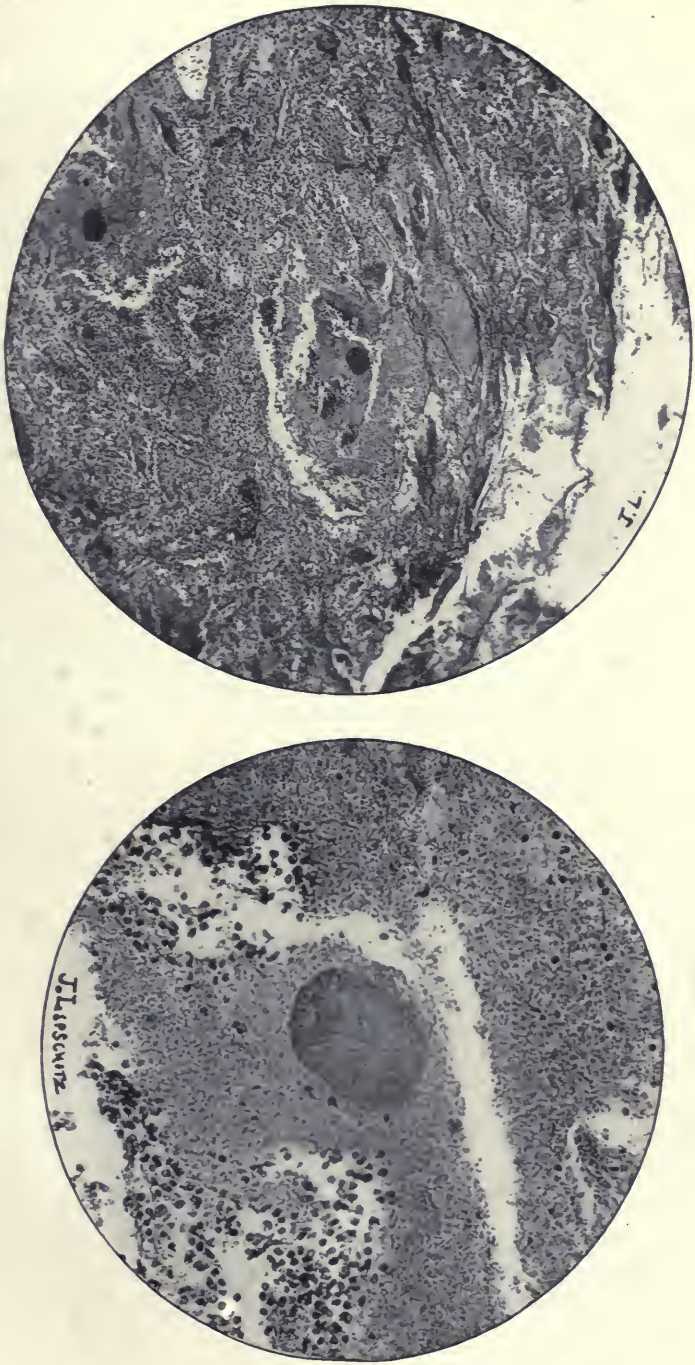
The urinalyses were negative.

A roentgenogram disclosed a dense white shadow occupying the upper three-fourths of the right pulmonary field, which was interpreted as fluid—perhaps a lung abscess or a sacculated empyema.

Many specimens of sputum were examined microscopically by the members of the medical staff, but no "ray fungi" were found. (Lebedeff, Machinskit, and Sokoloff mention cases which were diagnosed from the sputum findings.) The reports in each instance in our case showed polynuclear leukocytes, elastic tissue, streptococci, and staphylococci. Macroscopically the sputum was greenish yellow, thick, semisolid, tenacious, and foul-smelling. The odor was characteristic of gangrene of the lung and accordingly the case was regarded as extensive pulmonary gangrene, the cause of which was not evident.

Operative Procedures.—In view of the physical signs, clinical course, and roentgenographic findings, the chest was aspirated in the sixth right interspace in the midaxillary line. After several attempts with needles of increasing caliber, 8 c.c. of creamy greenish yellow, tenacious and very foul-smelling pus were evacuated. This was later reported on fresh smear examination to contain staphylococcic and Gram-positive diplococci. Culturally there was no growth but some anaerobic bacteria were present.

The thoracotomy, preceded by an exploratory aspiration, the needle being left *in situ* as a guide, was performed the same evening under nitrous oxid and oxygen anesthesia. The site chosen was in the same area from which the pus had been previously obtained. A pint of thick creamy pus was slowly evacuated. The work had to be done rapidly as the condition of the patient even while on the operating-table, was precarious. A large rubber tube was quickly inserted, the dressing applied and the child taken to the ward.



Figs. 2 and 3.—Actinomycosis pulmonis (primary).

His condition continued serious with attacks of restlessness, delirium and stupor lasting a week, the patient dying from toxemic paralyses and exhaustion.

The history of trauma to the chest and the obscure etiology of the pulmonary gangrene in an apparently well-developed boy aroused great interest; there was also a strong suspicion that external violence had been a contributory factor. The condition was therefore reported to the coroner's office in the hope that we might perhaps get an opportunity through necropsy to ascertain, if possible, the cause of this illness. A post-mortem examination was performed by Dr. Otto Schultz, who, after having seen gross sections of the gangrenous right lung, suggested the possibility of actinomycosis; but not until a thorough search for other foci was made (an important point in the opinion of McKenty), did he consider the disease as of primary origin in the lungs. The diagnosis was later corroborated by stained microscopic sections. A detailed gross pathologic report of the condition in the lungs follows:

Necropsy Findings.—The right upper lobe is very large and completely solid. Its pleura is thickened and covered everywhere with fresh fibrin. On cross-section this lobe is almost completely gangrenous, green, and honeycombed with abscesses containing yellowish-green pus, the largest of them being the size of a hen's egg. These abscesses are not encapsulated, but are lined by dirty irregular shreds of lung tissue. The lower lobe is studded with numerous patches of bronchopneumonia and from the center of the majority of these patches small amounts of pus can on pressure be forced out. The remainder of the lung is solid with reddish-gray hepatization. The left lung is covered with a thin layer of fibrin on its outer surface. This, on section, shows numerous patches of red hepatization which are wide-spread. The bronchial lymph-nodes are larger and softer than normal.

The microscopic examination by Dr. Eli Moschcowitz, pathologist to the Beth Israel Hospital, gives the following condensed report:

The pulmonary alveoli are completely filled with exudate, in part consisting of almost pure fibrin and in part with fibrin and large round mononuclear cells. Scattered throughout the consolidated lung are discrete darker areas which under low power resemble miliary abscesses. These areas vary in size from the head of a pin to a millet-seed and consist of collections of small round cells in the center of which are "ray fungi" varying in number from one to a dozen. The lung tissue in these areas is absent, the wall consisting of partially destroyed consolidated alveoli. The exudate immediately surrounding the "ray fungi" is completely necrotic. The walls of the lung alveoli are normal. The pleura is covered by a thin layer of hyalinized fibrin.

In reviewing rather hastily the more recent literature, we find the disease to be rare in children. In McKenty's series of thirty-seven cases seen in America (nine in females) occurring within 100 miles of Montreal, the youngest patient was a boy, aged 10. The incubation period varies from a few days in the cases occurring in the jaw to weeks and months in the abdominal and pulmonary varieties. It has been found, as a result of observation and experiments, that infection does not occur unless there is a lesion in the skin or mucous membrane.

Foulerton lays stress on the fact that, in many cases, an exact diagnosis on clinical evidence is impossible until the chest or abdominal walls are involved by extension of the process outward. Perforation of the skin then occurs and the organism may be discovered in the secretions on microscopic examination.

Incidentally we may refer to the disappointment in not being able to find or isolate the organism in the sputum or other secretions in some instances. The difficulties in the positive identification of the parasite by laboratory methods may be due to several causes: in the first place there is extreme difficulty of obtaining growth of many parasites of this class on artificial mediums, and, secondly, because of the varying morphology at different phases of the life-cycle of the parasite, recognition is not easy. It is well known that the mycelial or "ray-fungus" form represents only one of the three forms in which the parasite occurs. The typical mycelium and the mycelium in the earlier stages of "fragmentation" when it is breaking up into "rod forms," represent the only stage at which the parasite can be recognized positively by the microscope. At a later stage when sporulation has occurred, the irregular "bacillary" form and spherical spores resembling the pus cocci are seen, both staining deeply by the Gram method.

281 East Broadway.

IN MEMORIAM THOMAS MORGAN ROTCH, CHARLES PICKERING PUTNAM, FREDERICK FORCHHEIMER

THOMAS MORGAN ROTCH

Rotch wrote in 1873, "The Emigration of the White Corpuscle in Inflammation," an essay to which was awarded the first prize of the Boylston Medical Society for 1873; and in 1878, "Absence of Resonance in the Fifth Right Intercostal Space, Diagnostic of Pericardial Effusion," for the Massachusetts Medical Society.

The first impressed me as the work of a young man—he was born Dec. 9, 1849—given to study and scientific ambition; the second told of good observation and practical tact. That is why I invited him to act as secretary to the Pediatric Section of the American Medical Association which was to be founded. Never was a place accepted with more eagerness and filled with more capacity and success.

The meeting of the American Medical Association of 1880 took place at Richmond, Va. In that year and at that place there was founded the Section on Diseases of Children which has existed these thirty-four years with increased vigor and usefulness. Twenty years previously a futile attempt had been made to establish such a section in the New York Academy of Medicine. A second attempt was made in 1885, which has proved a success. This American Pediatric Society of yours has existed since 1888. You all know that pediatrics, since the second president, J. Lewis Smith, and Thomas M. Rotch, the third, has evolved into a powerful and vigorous body, nor are you unacquainted with the great influence Dr. Thomas M. Rotch has wielded in the development and fertility of pediatrics in American Medicine, which had to overcome more obstacles in this country of ours than in any country on the globe with the exception of Great Britain. Reactionary powers, such as antivivisectionism, antivaccinationism, sectarianism, and the enemies of autopsies, together with the lack of thoughtfulness and of perfect and uniform education in professional and well-to-do circles, such as those of clergymen, lawyers and legislators, have combined to cripple or obstruct the science and art of medicine in the United States.

After his return in 1876 from Europe where he studied in Berlin, Vienna and Heidelberg, he practiced in Boston, always with a view to the welfare of children and the teaching of their physiology and diseases. The pediatric department of Harvard University owes him everything. That university was almost the first in America to establish a special chair for pediatrics as the proper reward for his incessant labors in that branch of medicine. It has happened in connection with that fact, and it is characteristic of his modesty, that Rotch asserted that the administration of Harvard was induced to raise that chair to a full professorship only by the appreciative mention in the introduction to the first volume of Keating's Cyclopedia of Pediatrics, of pediatric work and the necessity of establishing greater facilities for the study of the diseases of the young.

In this movement, Harvard was preceded by one medical school only. That was a small "New York Medical College" in East Thirteenth Street, New York. Its reorganization in 1860 included a regular professorship for the diseases of children, and created the first regular general bedside instruction under its own roof. Both of these advancements died (but should not be forgotten in the history of American medical education) when the school had to be closed in the last year of the Civil War.

The Archives of Pediatrics of 1887 and the medical publications of the Harvard Medical School of the same year contained a paper of Rotch on "The Artificial Feeding of Infants." One on "Breast Milk" was published in 1890, a "Presidential Address" in 1891. They were followed by a paper on milk laboratories in 1893, on substitute feeding in 1896, on modified milk in 1897, on the history of percentage feeding, another on pasteurization of milk for public sale, a third one on Roentgen ray in 1907. Meanwhile, since 1896, he published successive editions of his large book which he called "Pediatrics."

His labor was not limited to the study of diet. He wrote on acute exanthems in 1897, on the perforation of the stomach in an infant 7 weeks old, in 1899, on rachischisis in 1900, on infantile scorbutus, on the study of pediatrics, on tuberculous peritonitis in 1903, on diarrhea in 1904, on gonorrhea and syphilis in 1907.

Nor was he working alone; his endeavor was often to have others, usually young men, cooperate with him. He published with Cushing, decapsulation of both kidneys; with Dunn, pulmonary or osteoarthropathy; with Floyd, opsonic index and tuberculin test; with George,

normal living anatomy, and osteomyelitis; with Holt, nomenclature of diseases of the gastro-enteric tract; with Ladd, pernicious anemia; with Low, blood cultures; with Murphy, gastro-enteric obstruction.

Dr. Rotch's labors and activities were so many and so successful as to render his positions and places of honor and responsibility insignificant. He was one of those who are counted by what they do and not by what people say of them. Still his honors were many and more than what J. McKeen Cattell enumerates in his "American Men of Science." The following lines refer to Rotch:

A.B., Harvard, 1870; professor pediatrics, Harvard, 1893; consulting physician Boston City Hospital; visiting physician Children's Hospital; medical director Infant Hospital; consulting physician Infant Hospital, London, Eng.; Fellow of the Association of American Physicians, of the American Pediatric Society (once its president), American Medical Association, Massachusetts Medical Society, Suffold District Medical Society (once its president). Let this, however, suffice.

Dr. Rotch is one of the men whose greatness is won by hard work in a limited sphere. He appreciated the boundary lines restricting everybody, but he felt from the beginning of his career the necessity of constructive work. That is why a large number of his papers are dedicated to the subjects of infant feeding, and particularly percentage feeding. His call to present to the British Medical Association the methods of what was considered the American method of infant feeding, was the first proof of the impression his personality and searching had made far and wide, and the foundation, in London, of a milk laboratory like that established by Rotch in Boston. It is well known to you all that the first Boston Laboratory was not installed in America until Rotch had proved to the profession in New York and other cities the desirability, or rather indispensability, of clean milk of high-grade for infant feeding. That example became the teacher both of the profession and the public, aye, even the trade, whose interest is believed by many to be best served by the selling of the worst possible article for the highest possible price, and whose virtues are often considered commensurate with the dangers or fears of punitive measures. Nobody knew that better than Rotch; that is why he never failed in his suspecting watchfulness in connection with milk laboratories.

I now quote a line from Dr. Dunn's review in the Boston Medical and Surgical Journal:

"For the last twenty years Professor Rotch was much interested in developing the work of the Infant Hospital, the first hospital in this country to admit patients restricted to the first two years of life."

This is only part of the initiative work of Dr. Rotch. It must be left to those who are intimate with his daily life and exertions to record in appropriate terms and with sufficient energy the great merits of Rotch in connection with his searching and working in the two large hospitals he controlled. Now the number of constructive physicians in this country is not large. Not one of them should be buried in silence; least of them a man who has genuine and permanent merits. These were his vast knowledge, enthusiastic industry, clear and logical expression and other gifts of a great clinical teacher. These gifts were increasingly developed with every year of his teaching life. The disproportioned chapters of his great "Pediatrics" were improved from edition to edition. The first of them, to a large extent, indeed, one-fourth of the book, filled with the feeding of infants. Gradually it became a hand-book of greater usefulness and proper proportions. With years and learning, guided by his watchful brain and warm heart, he became more statesmanlike in his views and researches. His big book, "The Living Anatomy and Pathology of Early Life as Shown by the Roentgen Method," was pervaded by a humane and humanistic study of the growing child. He gave a distinct discrimination between the actual and chronological age of the growing child, insisting on the cautious examination of a child wishing or compelled to enter on manual labor, according to the anatomic development of the body as exhibited by his osseous growth. Rotch worked assiduously and successfully for years, giving credit to writers who had worked in the same direction. It is exactly that class of research and that class of students that will prove the blessing of the cooperation of science, sociology and conscientiousness.

It is quite possible that Rotch will be mostly remembered by the permanent influence his hospitals will exert on the health of thousands of patients, and on the ever-living schooling which students and doctors will derive from his creations. This influence I estimate much higher than the immediate good done to the sick. This value to the profession is greater than that which would accrue from similar work performed by men equally gifted or more personally ambitious. Dr.

Rotch was a general practitioner, a great searcher, and to all his work he added the outflow of his kind and humane heart and the appreciation of the fact that only a good man can be a great and good doctor. That is what Hippocrates meant when he said that where there is love of our calling there is love of mankind, or Nothnagel when he said before closing his eyes: "Only a good man will be a good doctor." That *is* what Rotch *did*. Though being given to accurate research he never severed it from the dictates of conscience and humanity. No medical man who does research work alone, laboratory work on dead tissue alone, will ever rival in his results, the warm-hearted person who studies the soul of the patient who is under observation, and estimates the innermost folds of a man's heart who clamors for relief. The human suffering body is to a man like Rotch much more than his physiologic make-up.

There is still more to Rotch's credit. There are those who disagreed with him in regard to his theories and teaching on the feeding of babies. Those here and outside who find fault with him should not forget what they inherited from him before he died. Our inheritance is the impressive teaching of the necessity of studying the infant and child. He belongs to the few who studied and taught pediatrics because they could not help it. No distress of his own, stoutly borne because fate brought it, made him shirk or interrupt the hard work required to accomplish his beneficial ends. Let nobody forget that it was he amongst a few who in America raised pediatrics to the rank of a genuine science and humane practice. I wish my American colleagues would soon appreciate the fact that the young men you send to certain European centers to learn medical science and morals, find their goal more often after their return, when they meet the good and great men represented in this and other scientific bodies.

A. JACOBI.

CHARLES PICKERING PUTNAM

Dr. Charles Pickering Putnam, for many years a member of this Society and its president in 1909, died April 23 last, in his seventieth year. He was one of a family of physicians, his father having been Dr. Charles Gideon Putnam, and his grandfather, Dr. James Jackson. He graduated from Harvard College in 1865 and from the Harvard

Medical School in 1869. After studying for a time in Germany, he began the practice of his profession in Boston in 1871. He was a general practitioner, but for many years made a specialty of pediatrics. He was a lecturer on Diseases of Children at the Harvard Medical School from 1873-1875, and a clinical instructor in the same subject until 1880. At this time he also did some excellent pioneer work in orthopedics.

He served the Boston Dispensary as district physician from 1871 to 1873, and as orthopedic surgeon from 1873 to 1876. He became physician to the Massachusetts Infant Asylum in 1875, and was also president of its board of trustees from 1898 to 1910. He was chiefly instrumental in the development of the boarding-out system established by this hospital many years ago.

Dr. Putnam was from the very beginning of his career the leader in Boston in charitable and social work connected with children. He was one of the founders, in 1873, of the Boston Society for the Relief of Destitute Mothers and Infants, which was a pioneer in establishing the policy of keeping mother and child together. He was president of this society from 1904 until his death. He was one of those who, in 1879, took part in the movement for establishing the Associated Charities of Boston, the second charity organization society in this country. He always took a very active part in its work, and was its president since 1907.

Dr. Putnam took a leading part for many years in all the movements for the care of the poor and of the neglected and delinquent children of Boston. He held many public positions, among the most important of which was that of chairman of the Board of Children's Institutions, from 1902 to 1911. His work was never properly appreciated and he was frequently misjudged, largely because the public and the politicians were unable to understand the absolute honesty of the man and to realize that he was always working for the good of the community and not for his own advantage or preferment.

He was one of the incorporators of the Boston Medical Library, in 1875, and served on important committees of this institution until his death. He helped to organize and carry on the Directory for Nurses established by this institution and at the time of his death was in charge of it. This was the first directory for nurses established in Boston.

He was a tireless worker, and a remarkable organizer. He had, in addition, the unusual faculty of interesting others and of making them work as hard as he did himself.

He was the ideal family physician, beloved by all his patients. No one can ever quite fill his place for them. No one man can take his place in the community and in public work. It will take many men to do that.

JOHN LOVETT MORSE,
CHARLES HUNTER DUNN.

FREDERICK FORCHHEIMER

"And so he dwelt among men, physician and sage. He served them, loving them, healing them, sick or maimed."

Truly this was a life of loving service; and surely there came the reward: he was the beloved physician.

Frederick Forchheimer was a man of unusual attainments, and of noteworthy achievements. A great clinician with all that it implies, a profound scholar, a wise teacher, he was; and with it all a man of wondrous charm. He had to a most uncommon degree the quality of personal magnetism which drew to him countless friends and held them fast. To know him well was to love him; and to have his affection was to have that which made its possessor proud. His was a busy life, and yet there was always time for kindness—in thought and deed—for those who needed him.

He was one of the rare souls who found his greatest pleasure in the happiness he could bring to others.

An eminently successful practitioner, he remained always an idealist in medicine.

His influence in the lay world was tremendous—and always for good. His position in the profession was that of earnest student, of painstaking teacher, of wise counselor to whom all might turn for aid.

One of the founders of this Society, one of the pioneer teachers of pediatrics in this country, he retained always his special interest in this branch of his work. His was the broad outlook, and his the comprehensive grasp. The exactions of an arduous practice left him little time for purely research work in the later years of his life, but his interest in scientific medicine was ever keen, his knowledge of its attainments always sure.

To the medical world at large Frederick Forchheimer was known and favorably known, by his books.

To the multitude to whom he gave his medical skill, ever unstintingly, ever unselfishly, he came as a carrier of cheer and helpfulness.

And to those—and they are many—who were privileged to call him friend, he was an ever-present inspiration. The rewards of his well-spent life were many. Not the least of them was the love that came to him from all who knew him.

B. K. RACHFORD,
C. G. JENNINGS,
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